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DIRECT VERSUS INTERMEDIATE PATHWAYS IN INFECTIONS OF THE MASTOID

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The present study was undertaken to determine, if possible, the most common pathways by which ordinary pyogenic infection of the upper respiratory tract may reach the middle ear and the mastoid process.

Despite the common idea (prevailing hypothesis) that involvement of the middle ear and the mastoid is caused by the direct extension of infection from the nasopharynx through the eustachian tube ("the normal anatomic pathway"), we have collected sufficient evidence to consider the blood and lymph channels as the more direct and common pathways for such infections.

The hypothesis of direct extension, in our view, does not adequately account for the fact that cultures of material from the nasopharynx will invariably grow streptococci although the early serosanguineous transudate from the middle ear is sterile. Similarly, the hypothesis does not explain the presence of isolated areas of broken-down mastoid cells, usually those about the sinuses, without the presence of pus in the mastoid antrum and its adjacent cells, because, if the process were in fact one of direct extension, then the cells of the antrum would necessarily become involved before the more distant cells of the mastoid. Furthermore, in the early stages of otitis media, there is no adequate explanation for the tenderness over the mastoid process when the only positive roentgenologic finding, a cloudy appearance of the air spaces of the mastoid, points to inflammation of the mucosa of the mastoid cells.

It is possible that the serosanguineous transudate found in the middle ear and in the mastoid spaces early in the course of the infection is sufficiently bacteriostatic or even bactericidal to account for the negative cultures. Nevertheless, we have satisfactory evidence for suspecting that the progress of infection in the tympanum and the mastoid is from within toward the periphery, i. e., from the submucosal

A preliminary report from the Department of Otolaryngology, Beth Israel Hospital (Dr. S. J. Kopetzky). Further studies are in progress.

tissues to the air spaces and not from the epithelial lining of the same cavities to the subepithelial layers.

To guide us in determining the actual pathways of propagation, we have attempted a direct approach to the problem, one which requires the particular localization of bacteria in the tissues and the actual demonstration of their presence, dead or alive.

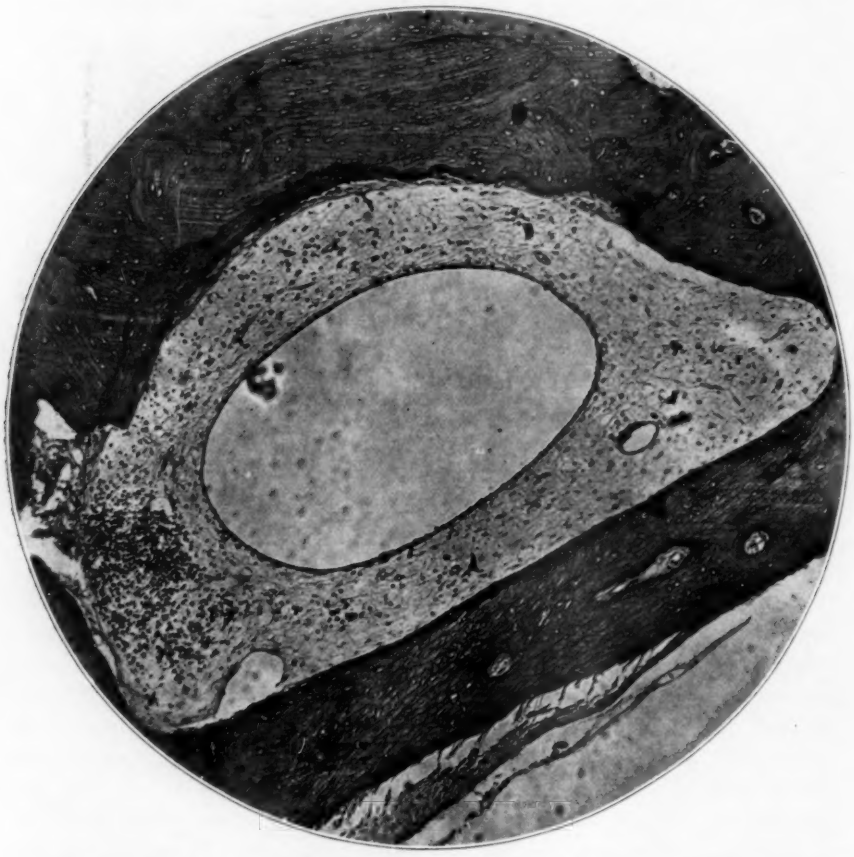

Fig. 1.—Figures 1 to 4 show the stages of inflammatory reactions in the air cells of the mastoid.

The early inflammatory phase is illustrated in this figure. This phase includes: (*a*) Filling of the air space with a serous transudate. As has been stated in the text, no bacteria can be demonstrated in the serous transudate. (*b*) *A* change from the normal squamous epithelial lining into a low cuboidal type. (*c*) Edema of the submucosal tissues, due in all probability to a toxic tissue reaction (for explanation consult the text). (*d*) Dilatation of submucosal vessels and beginning endovascular and perivascular changes. At the lower left corner one notices the beginning of a focal perivascular infiltration with round cells. Pus cells at this stage are usually very few.

The greatest difficulty in our early studies was the determination of a method whereby the streptococci could easily be demonstrated in the stained tissue sections. We have reason to believe that bacterial growth in living tissue is not similar to growth in artificial mediums. Bacterial morphology as we are accustomed to see it in artificial mediums is encountered rarely in living tissue, particularly in the case

Fig. 2.—The later inflammatory phase. The following changes are characteristic: (*a*) The serous exudate illustrated in figure 1 is now replaced by a formed exudate consisting of erythrocytes, pus cells and other tissue cells in a network of organized fibrin adhering to the epithelium in places. The source of this exudate was the hemorrhage from the necrotic focus *F* in the submucosa, which eventually became an organized blood clot. (*b*) The continuity of the epithelial lining is broken at *F*, indicating that the necrotic process has eroded this part of the adjacent mucosal lining. (*c*) The submucosa shows persistence of edema, dilatation of blood vessels, further progress of endovascular and perivascular changes and an increase in the number of focal perivascular infiltrations. (*d*) The perivascular infiltration at *F* has progressed to the stage at which necrosis and erosion of the epithelial lining have taken place, causing a hemorrhage and subsequent infection of the cell space of the mastoid.

of streptococci. In sections of tissue streptococci may occur either singly or in pairs, and owing to their altered morphology it often becomes difficult to decide whether they are organisms, fragments or broken-down tissue cells, artefacts or even precipitates from the stain. Unless they are grown to full maturity and acquire their familiar morphologic characteristics, there is no sure way of determining their origin. Therefore, in attempting to demonstrate the presence of strep-

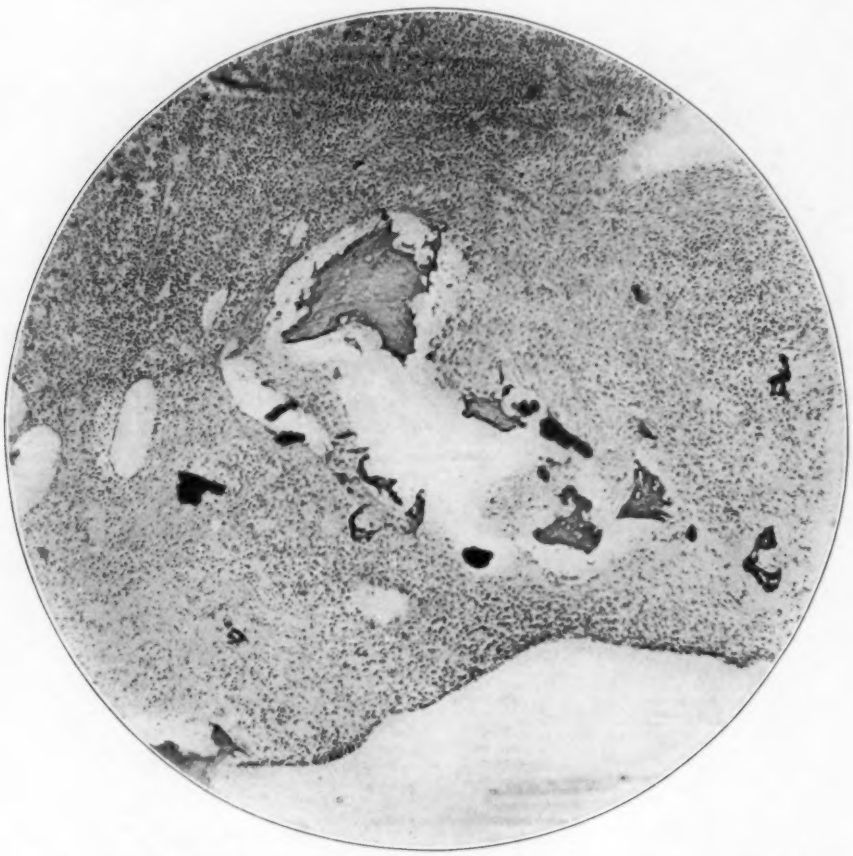

Fig. 3.—The final inflammatory phase. The structural outline of the air cell is lost. The remains of this destructive process are now found in the granulation tissue, which temporarily replaces the destroyed air cell.

tococci in mastoid tissue, we resorted to Dr. Eugene Frankel's technic, one which enabled him to demonstrate Bacillus typhosus in cultured biopsy material from roseola typhosa. Our modification of his original technic took the following form:

The fragments of mastoid tissue removed at operation are immediately placed in 10 cc. of nutrient broth of p_H 7.4. After from three to four days' incubation

at 35 to 37 C. the specimens are washed in distilled water and fixed in a 10 per cent dilution of solution of formaldehyde U. S. P. The cultured tissues then are dehydrated, embedded in paraffin and sectioned as usual. With sections 4 microns thick there is no difficulty in studying the morphology and arrangement of bacteria. The staining is by the ordinary Gram method, with the one variation that decolorization is done in acetone instead of in absolute alcohol. One short, quick washing

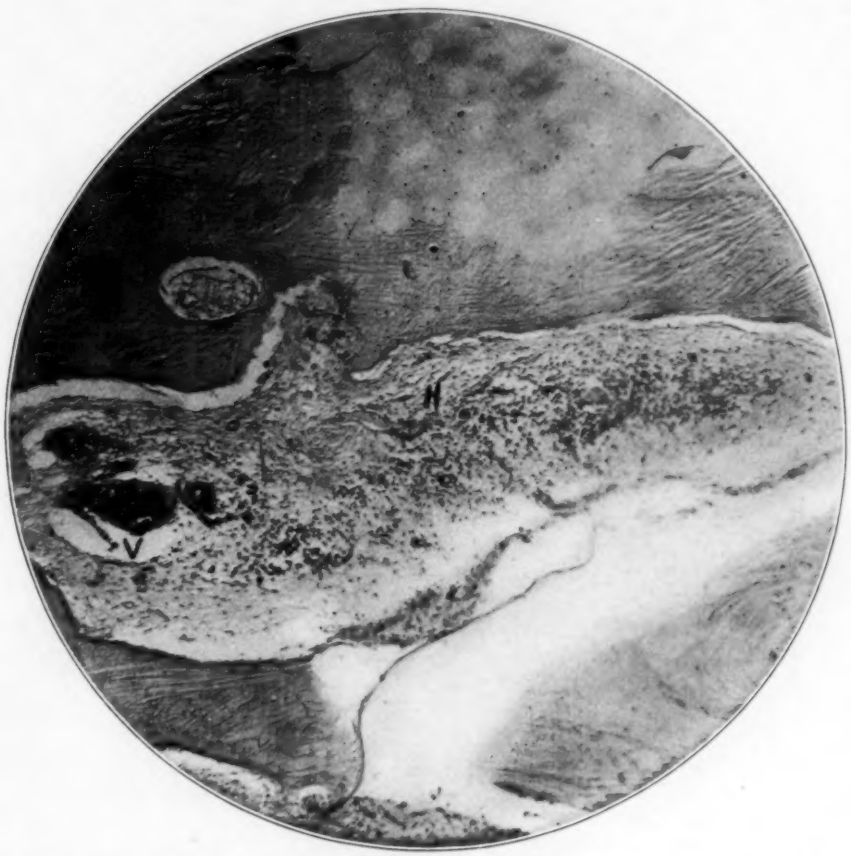

Fig. 4.—A demonstration of the endovascular and perivascular infiltration in a thrombosed vessel of the mastoid process. At *V* the thrombosed vessel is cut vertically and at *H* horizontally. The infiltration in this figure is mostly of the polymorphonuclear type.

in acetone is sufficient to clear the tissues of all precipitates from the stain while the bacteria still retain the stain.

To assure the success of the technic and the reliability of findings, it is of utmost importance to avoid all possibilities of extraneous contamination. With the introduction of but a few motile bacilli into the cultures, the results would be not merely valueless but misleading.

Once a motile contaminant gains access to the cultures, it will invariably invade the whole tissue, as already demonstrated.[1] On the other hand, similar experiments conducted with nonmotile organisms (the group comprising micrococci, staphylococci, streptococci, pneumococci, etc.) proved the inability of such organisms to penetrate the cultured tissues beyond their exposed surfaces.[1]

Fig. 5.—Streptococci in tissue spaces, probably lymphatics. In twelve photomicrographs (figs. 5-12) an attempt is made to demonstrate the presence of the infective agent (the streptococcus) in the various tissues in order to determine which are the most common pathways of infection of the mastoid. In all these photomicrographs, which are mostly low power reproductions, the intensely dark-staining bodies are masses of streptococci. Figure 8 is an oil immersion magnification of one of them to show the characteristic morphology of the streptococcus.

1. Hadjopoulos, L. G., and Burbank, Reginald: J. Bone & Joint Surg. **15**: 471, 1932.

According to the researches of Tillett and Garner,[2] which since have been substantiated,[3] the majority of hemolytic streptococci are capable of producing a fibrinolytic ferment. From these findings it might be expected that the effects of this lytic ferment may prepare the way for streptococcic invasion of deeper tissues. Our experience with experimentally inoculated sterile tissue cultures was not in accord with this

Fig. 6.—Streptococci filling a tissue space, probably a lymphatic space.

view. In the presence of fibrinolysin in our cultures, the microbic penetration could not extend beyond the exposed surface area of the tissues. If streptococci could indeed penetrate tissues indiscriminately, they should be found scattered more or less uniformly throughout the tissue

2. Tillett, W. S., and Garner, R. L.: J. Exper. Med. **58**:485, 1933. Tillett, W. S.: J. Bact. **29**:111, 1935.

3. Neter, E.: Proc. Soc. Exper. Biol. & Med. **34**:735, 1936. Neter, E., and Witebsky, E.: ibid. **34**:549, 1936.

network, as in the case of motile bacilli. Instead, as this report presently will show, the streptococci were found to be localized strictly in certain channels in the tissue, some of which were blood vessels, others lymph channels and still others plain tissue spaces of indeterminate nature.

Because tissues removed from the body have no circulation, and since nonmotile organisms deprived of circulation will grow only by direct

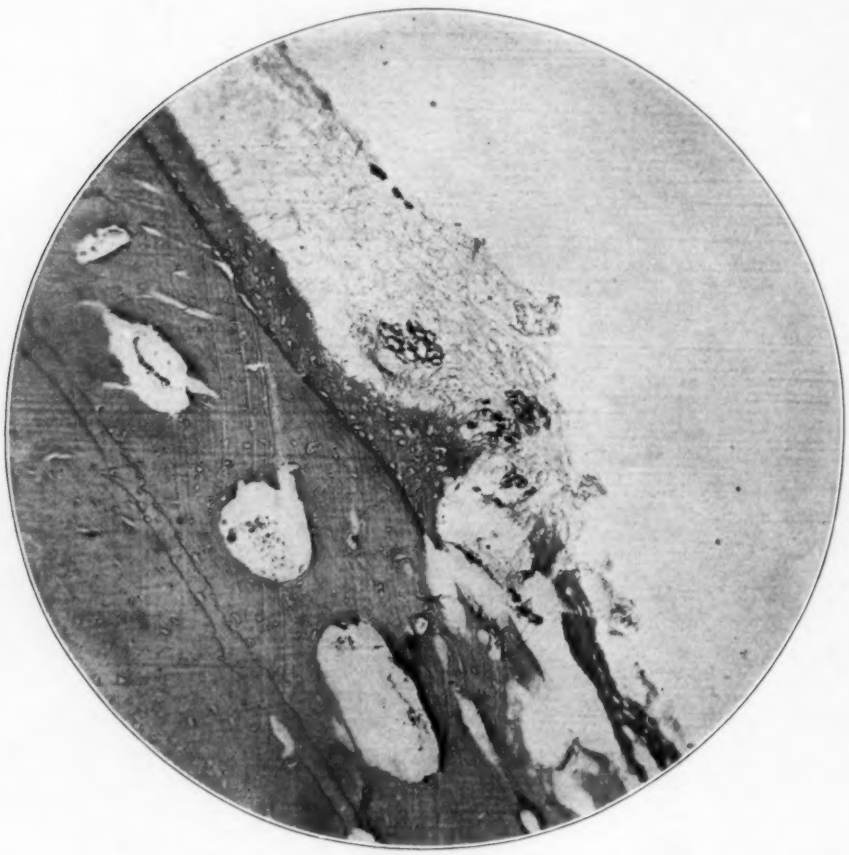

Fig. 7.—Streptococci in tissue spaces (lymphatics?) in the periosteum of the cortex over the mastoid bone.

extension due to multiplication of their number, it must follow that the organisms found in the channels in our tissue must have existed in their several foci at the time the tissues were severed from the body. Evidently their original number was so small—perhaps a single coccus or diplococcus—that they would escape detection in ordinary stained tissues. However, when these same tissues were subjected to culture, these few cocci, released from the growth-checking effects of the reac-

tive forces in the living body, were able to grow to their full capacity until they filled the channels in question, thus rendering their identification easy. As will be observed in figures 1, 2 and 4, the endothelial lining of the vessels within such areas of tissue is thickened and the perivascular spaces are infiltrated with lymphatic and plasma cells. This is additional, although indirect, evidence that the streptococci

Fig. 8.—Streptococci in areas adjacent to the epithelium and also attached to the eroded surfaces of the epithelial lining.

encountered in our sections of tissue were present in these identical areas before they were inserted into the culture mediums.

We now proceed to the interpretation of the reaction of invaded tissue to the infective agent from the inception of the infection in the nasopharyngeal orifice of the eustachian tube to the production of mastoiditis and its sequelae. The first defense against invasion of the natural anatomic channels—eustachian tube to tympanum to mastoid

—will be found in the ciliary activity of the epithelial lining of the eustachian tube. If the disease is not aborted at this stage, the ciliary activity is paralyzed by the diffusion of toxins into deeper structures. The second line of defense is the resulting edema of the mucosa and submucosa in order to dilute the toxins and thus render them less harmful. To state whether edematous fluid has additional antitoxic

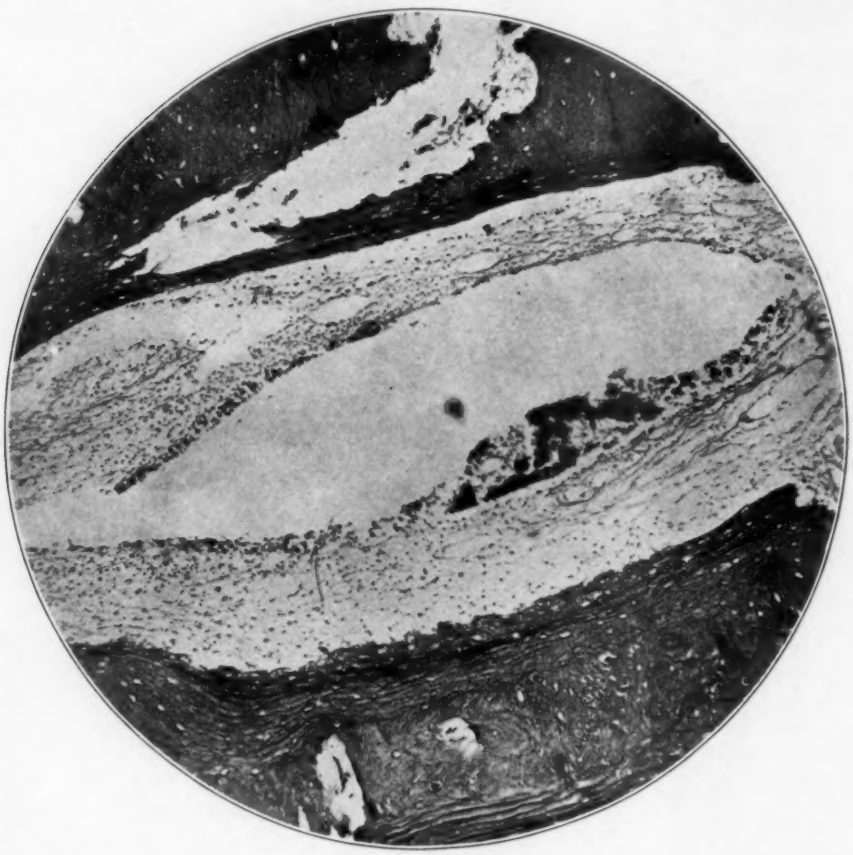

Fig. 9.—Streptococci within the exudate and attached to the epithelium of a mastoid air cell.

and antibacterial properties is not within the scope of this report. The edema in question, however, performs the function of disrupting the continuity of the anatomic channel, especially at the constricted middle portion of the eustachian tube. At this stage the tympanic membrane appears red and somewhat retracted and the tympanic cavity contains a small amount of serosanguineous fluid which, both culturally and by direct smear, can be shown to be sterile. The retraction of the drum

membrane is a result of the negative pressure in the middle ear which follows occlusion of the eustachian tube at its constricted middle portion, owing to the edematous swelling of the tubal mucosa.

Although there are definite inflammatory signs in the middle ear at this stage in the disease, one fails to find an infective agent. Being devoid of motility, streptococci and other micrococci, which are the

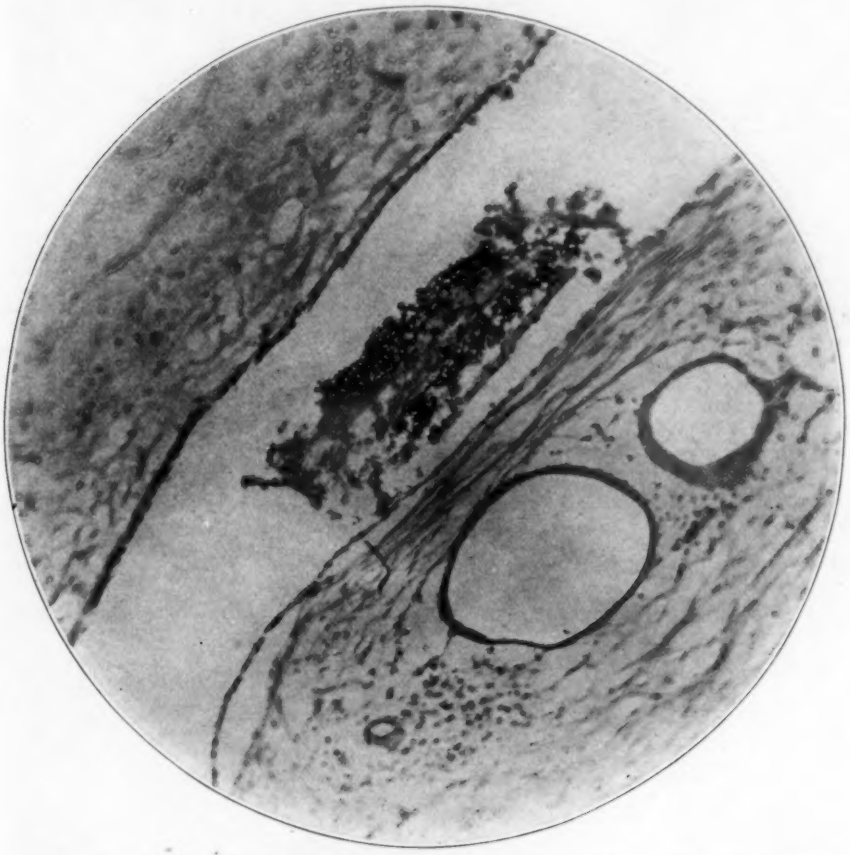

Fig. 10.—Streptococci within an exudate. For a description of the formation of an infected exudate consult the text and figure 2.

usual causes of infection of the mastoid, have to depend directly on propagation to invade the mucosal and submucosal structures, provided that these have been sufficiently devitalized by the diffusion of toxins. Partial or incomplete dilution or neutralization of toxins will ultimately result in small foci of necrosis, a good culture medium for further penetration of micro-organisms.

The anatomic location of these foci of necrosis will eventually determine the routes of the further propagation of the disease. If they are superficially situated in the mucosa, they may enter the natural anatomic pathways beyond the constricted portion of the eustachian tube by ulcerating through the mucosa (fig. 2) and draining into the eustachian tube, the tympanum or the air spaces of the mastoid (figs. 8

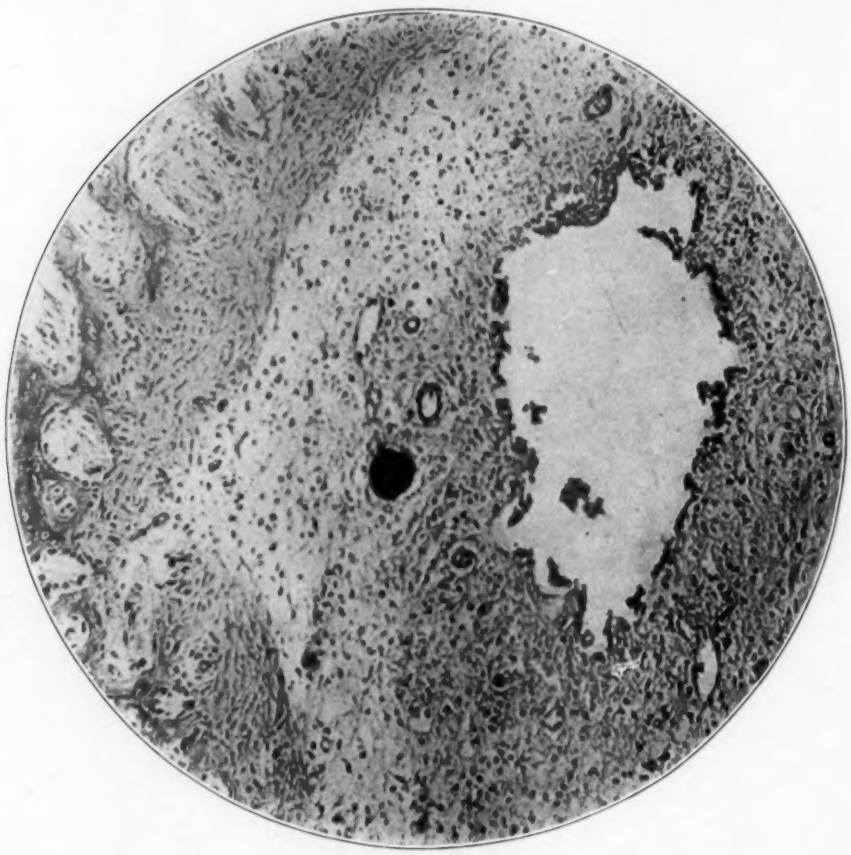

Fig. 11.—Streptococci filling the lumen and invading the walls of a submucosal vein. Figure 12 is an oil immersion magnification of this area.

and 9). This is what occurs in the majority of cases in which recovery takes place. When progression of the infection does ensue, other elements enter into the picture. The infection now will involve the capillaries and lymphatics of the submucosa (figs. 11, 12, 13 and 14).

The invasion of the arterial limb of a capillary, with subsequent necrosis, will not result in septicemia. All it can do is give rise to a small punctiform hemorrhage in the necrotic spot, but the outward

pressure of the arterial blood stream opposes the invasion of foreign organisms. Nevertheless, the arterial and venous limbs of the capillaries are so closely entwined that when one is involved the other is almost invariably involved also. An invasion of the blood stream, although it may be only temporary, thus cannot be avoided.

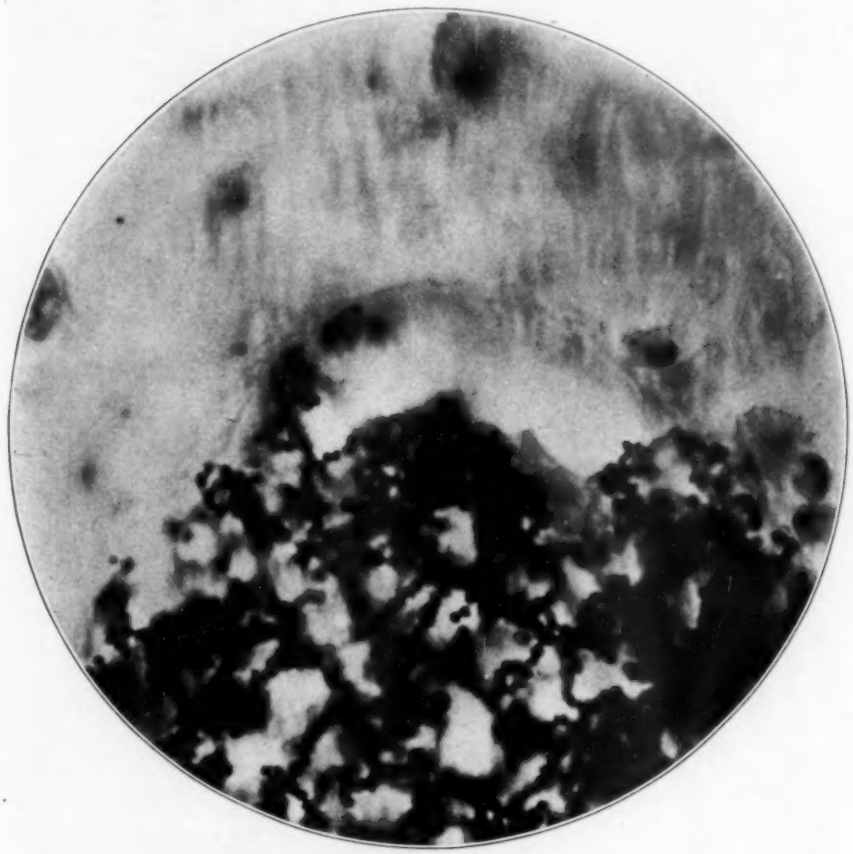

Fig. 12.—Oil immersion magnification of the infected vein shown in figure 11.

Under normal circumstances the blood carries a sufficient concentration of bactericidal bodies to take care of emergencies. The danger of fatal septicemia threatens only when larger vessels, the jugular vein, for instance, have been involved in the infective process. The lymphatics of the submucosa are similarly but more readily involved. At this stage of the disease the danger is not so much from septicemia as from the opening of new and more blood and lymph channels for the spread of the disease (figs. 4, 5, 6 and 10).

The involvement of the tympanic and the mastoid cavity is mainly through these channels and rarely, if ever, by direct extension through the eustachian tube. If the source of infection in the tympanic and the mastoid tissue were from within the spaces normally containing air, we should be able to demonstrate on our sections streptococci diffusely infiltrating the tissues. Instead, we have streptococci strictly limited to

Fig. 13.—Streptococci in a chain of vessels of the submucosa.

certain well defined areas in the mucosa and submucosa, of which some are definitely blood vessels, others probably lymphatic channels and the remainder indeterminate tissue spaces.

The presence of streptococci in the exudates filling the tympanic or the mastoid cavity (figs. 9 and 10) is not at all contradictory to our view, as such exudates may result either from rupture of a mucosal or a submucosal abscess into the cavity (fig. 2) or from a secondary infection of the exudate by infected leukocytic carriers (fig. 10).

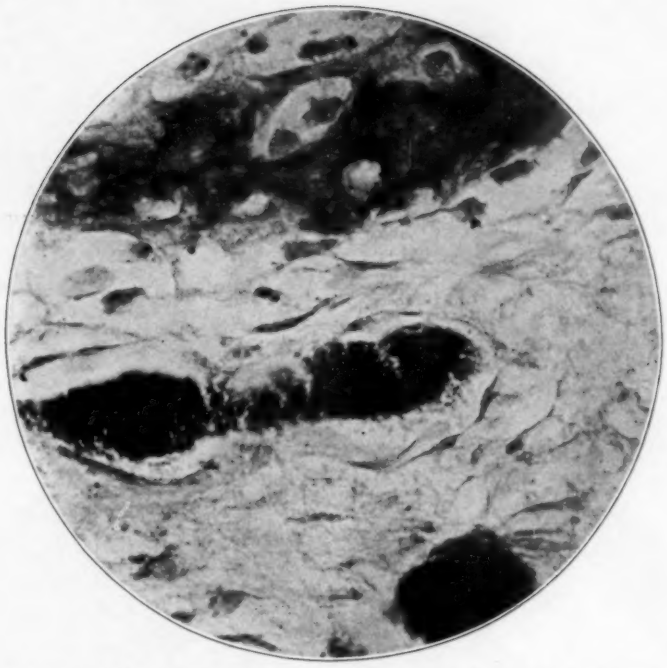

Fig. 14.—A high power view of the section illustrated in figure 13.

Fig. 15.—Streptococci in the lymphatics of the haversian canal.

In the case of larger vessels, such as the internal jugular vein (fig. 16), the source of infection is again through the capillaries and the lymphatics. When the jugular vein is involved by way of the capillaries, endophlebitis results. Because of this endophlebitis there are proliferation of endothelium, deposition of fibrin, slowing of the venous return flow and eventual thrombosis. If the progress of the infection is

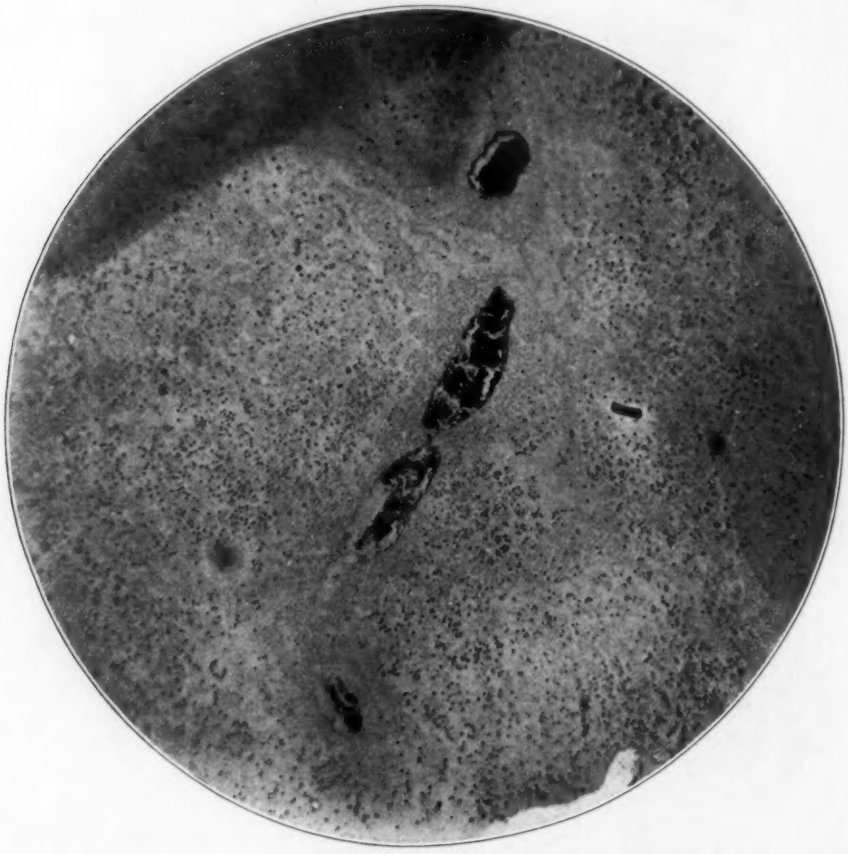

Fig. 16.—Streptococci within the secondary vessels of a thrombus of the internal jugular vein which has undergone secondary canalization.

unchecked, septicemia results, with its natural sequelae. When, on the other hand, the jugular vein is involved by way of the lymphatics, periphlebitis results. This periphlebitis may extend directly by way of the lymphatics along the petrosal veins to the cavernous sinuses. The lymphatics of the dura thus would be involved before the general blood stream, resulting in meningitis. Therefore, when the first complication of mastoiditis is septicemia in which the blood cultures are persistently

positive, one may conclude that the jugular vein has been involved by way of the capillaries. If, however, meningitis develops as the first complication, one may conclude that the infection has been transmitted by way of the lymphatics.

CONCLUSION

In contrast to the general belief that the common source of infection of the mastoid is through the natural anatomic atria—the eustachian tube, tympanum, antrum and mastoid cells—we bring evidence to support the less accepted theory that the major and more important sources of infection of the mastoid are the blood and lymph channels of the adjacent mucosa.

A means of demonstrating streptococci in mastoid tissues failed to show streptococci diffusely disseminated in such tissues. On the contrary, we encountered them strictly localized in certain channels in the tissues, of which some were definitely venous, others lymphatic and still others indefinite as to origin but definite in outline.

SOME OBSERVATIONS ON THE MANAGEMENT OF INFECTIONS OF THE BLOOD STREAM FROM MASTOIDITIS

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A review of the entire subject of otitic septicemia will not be attempted here, since noteworthy contributions have in recent years summarized the results from extensive series of such cases.[1] Certain points in clinical management of two recent cases are submitted briefly herewith for criticism and comment.

CASE 1.—A well-grown lad of 10 years retained his adenoids and the major part of each tonsil after an operation abroad at the age of 5 years. Three weeks after a mild attack of measles he complained of nasal obstruction and pain in one ear. Within eighteen hours the right drum was found to be bulging and covered by dark serous blebs, and myringotomy released a copious flow of reddish seropurulent exudate. The entire mastoid region was extremely tender, and roentgen films showed all cells filled, those at the tip being very large. The temperature on myringotomy was 101 F.; it dropped to normal and then rose, with slight chilly sensations, within six hours to 103.6 F. The white cell count was 16,000. There was no nuchal pain or choking of either optic disk.

An immediate and extensive simple operation revealed complete invasion of the mastoid by long chain hemolytic streptococci without breaking down of trabeculae or of the sinus plate, which was very thin. The sinus wall was normal in appearance and fulness; hence it was not opened. Within two days, however, the septic temperature continuing, blood for culture was taken on the rise and the sinus was widely opened, without evidence of obstruction or clot in either direction, and the right jugular vein was found similarly normal in appearance, but was tied at the same session. Blood culture revealed long chain hemolytic streptococci.

The usual paleness and slight cyanosis, with extreme prostration, semicoma and a temperature between 104 and 97 F. continued over a course of four weeks, with occasional chills at the start. Six blood transfusions, from 160 to 180 cc., were given immediately after the operation and on the second, fourth, seventh, twelfth and nineteenth days thereafter. Blood culture was negative on the seventh and the twelfth day, and recovery, while slow, was uneventful.

From the Department of Otolaryngology, University of Oregon Medical School.

Read at the Forty-Third Annual Meeting of the American Laryngological, Rhinological and Otological Society, Atlantic City, N. J., June 3, 1937.

1. Seydell, E. M.: Sinus Thrombosis, Ann. Otol., Rhin. & Laryng. **41**: 466, 1932. Lillie, H. I.: Suppuration of the Temporal Bone Accompanied by Infection in the Blood Stream: Clinical Study, Arch. Otolaryng. **18**:630 (Nov.) 1933. Dill, J. L., and Crowe, S. J.: Thrombosis of the Sigmoid or Lateral Sinus: Report of Thirty Cases, Arch. Surg. **29**:705 (Nov.) 1934. Meltzer, P. E.: Treatment of Thrombosis of the Lateral Sinus, Arch. Otolaryng. **22**:131 (Aug.) 1935.

The left ear became acutely involved six months later, after a cold; it discharged for a week after paracentesis, and shortly thereafter the tonsil and adenoid remnants were removed. At the time of writing this boy has gained 14 pounds (6.6 Kg.) since his first illness.

CASE 2.—A boy of 11 years was brought in some hundreds of miles, after being grievously sick for three weeks after spontaneous rupture and subsequent paracentesis of both ears, with red seropurulent copious discharge. The temperature on arrival was 99.2 F. The white cell count was only 6,100, with 78 per cent polymorphonuclears; the red cells numbered 3,800,000. There was slight tenderness over each mastoid; both were cloudy roentgenologically, and moderate choking of the left optic disk was present. Culture of blood removed on the rise of temperature on the next morning showed a strong growth of long chain hemolytic streptococci. The Queckenstedt test on arrival disclosed a marked rise in pressure, to 420 mm., on compression of the right jugular vein and only 70 mm. on the left. In spite of the absence of local tenderness or edema, this test was repeated the next day, with identical results. A transfusion of 150 cc. of blood was given, and in view of the positive blood culture and evidence of sinus occlusion, the left mastoid was opened extensively, disclosing little or no trabecular damage but generalized seropurulent infiltration. The sinus wall was normal, compressible and thin and refilled promptly from either end; it was not opened but was packed shut with iodoform gauze rolls. The jugular vein was tied immediately, no clots being found in the neck. Blood culture was negative on the seventh postoperative day. Four more transfusions, averaging 100 cc., at intervals of three days were sufficient to bring the temperature down to normal in eight days and to dry up both ears within twenty-four days. Subsequent convalescence was uneventful.

The first of these boys had so violent an invasion of the blood stream within forty-eight hours that six large transfusions over a course of six weeks were required to bring his temperature to normal and to secure a negative blood culture. The second lad, whose tremendous drop in resistance was evidenced by his lack of fever and leukocytic response, had a complete occlusion of one jugular vein, probably at the bulb, yet he required only five small transfusions and was out of the hospital in less than four weeks.

In neither case was there evidence of any inflammatory process in the mastoid portion of the sigmoid sinus, or in the exposed jugular vein when tied. Yet the entire mastoid process in each child was filled with thin seropus and black clots. The bony trabeculae were still hard; there was no macroscopic evidence of thrombosis of emissary veins in the operative area. It seemed wise to isolate any possible obstruction or mural clot in the region of the bulb by destroying the jugular vein below as well as the sigmoid sinus in the mastoid. It was not felt that anything would be gained by splitting the normal-looking sinus in the second case. As much time as possible was saved in both operations by the use of ethylene anesthesia and by having a second clean surgical team attack the jugular ligation the instant the mastoid wound was packed and sutured.

There was no roentgenographic preoperative evidence of anomalies of the sinus to explain the closed left jugular vein in the second case.[2]

Discussion of otogenic sepsis in many places still revolves about the question of destroying the infected venous channel and turning out all clots therein. This is all very well when one finds a necrotic sinus wall, with its lumen occluded both ways. Obviously, when this condition is present, one must fish out clots as far as the bulb and torcular if necessary to secure free bleeding. Few are rash enough nowadays to recommend the Grünert operation for exposure of the bulb in these grievously sick hemorrhagic patients. Ligation of the jugular vein, an easy enough procedure, is criticized as useless in the face of a positive blood culture. Nevertheless, one feels better if the possible direct avenue from the bulb or the petrosal veins to the heart is blocked.[3]

Chemotherapeutic injections have not hitherto seemed to me to offer much hope. A recent German report concerning the injection of a derivative of sulfanilamide [4] (prontosil) in a case of otogenic septicemia showed multiple pia-arachnoid and pericardial hemorrhages, without arrest of the process in the blood stream.[5] The sulfonamides seem to promote phagocytosis of hemolytic streptococci,[6] and experimentation at Johns Hopkins University [7] and information obtained from the Council on Pharmacy and Chemistry of the American Medical Association [8] indicate the possibility of the development of a compound with a much simplified formula and less potential danger. The sulfonamides seem to augment the phagocytic activity of the leukocytes rather than to destroy germs free in the blood stream, the vain

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2. Woodhall, B., and Seeds, A. E.: Cranial Venous Sinuses: Correlation Between Skull Markings and Roentgenograms of the Occipital Bone, Arch. Surg. **33**:866 (Nov.) 1936.

3. Kopetzky, S. J.: Acute and Chronic Otitis Media, Sinus Thrombosis and Suppuration of Petrous Pyramid, Arch. Otolaryng. **24**:516 (Oct.) 1936.

4. "Sulfanilamide" has recently been adopted by the Council on Pharmacy and Chemistry of the American Medical Association as the nonproprietary name for para-aminobenzene sulfonamide base, which has been called "prontylin" among other names. The term "prontosil" has been used to designate various derivatives. The one employed in the case cited here was the disodium salt of 4'-sulfamidophenyl-2-azo-7-acetylamino-1-hydroxynaphthalene-3,6-disulfonic acid.

5. Seiferth, in discussion on Die otogene Sepsis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **38**:119, 1935.

6. (*a*) Wanner, in discussion on Die otogene Sepsis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **38**:113, 1935. (*b*) Voss, in discussion on Die otogene Sepsis, ibid. **38**: 115, 1935.

7. Long, P. H., and Bliss, E. A.: Para-Aminobenzenesulfonamide and Its Derivatives: Clinical Observations on Their Use in Treatment of Infections Due to Beta Hemolytic Streptococci, Arch. Surg. **34**:351 (Feb.) 1937.

8. Leech, Paul N.: Personal communication to the author.

hope aroused a few years ago by the injection of various dyes. While sulfonamides, such as sulfanilamide, are not bactericidal in vitro, chemical change into an actively bactericidal substance seems to take place within the body.[6a] Intravenous injections of dyes, blood transfusions and the intramuscular injection of sulfonamides, which is now preferred to intravenous or oral administration,[7] all provoke a more or less febrile reaction, which should not cause alarm. Two infections of the blood stream by short chain beta hemolytic streptococci, in children aged 7 and 9, one arising from involvement of both mastoids and the other from subacute unilateral otitis media without involvement of the mastoids, have recently been under treatment by my associates. The child with both mastoids involved had five blood transfusions without effect on the temperature, which reached 106.6 F., with rapid loss of leukocytic resistance (5,500 polymorphonuclears) and evidence of focal involvement in both knees and one thigh. The child with otitis after one transfusion, with continued chills and fever, complained of pain in one knee, but the leukocytic response remained high (19,000). Intensive use of sulfonamides intramuscularly and by mouth has progressively brought down the temperature, stopped the chills and focal symptoms and improved the blood picture of both patients, who are still under observation at the time of writing and concerning whom further reports will be made after recovery is complete. Intravenous administration (10 cc. of a 2.5 per cent solution of the derivative of sulfanilamide just mentioned [prontosil]) was found to produce vomiting, but was required twice in the child with involvement of the mastoids. The intramuscular dose thereafter was 5 cc. of a 2.5 per cent solution every eight hours, with 5 grains (0.32 Gm.) of sulfanilamide by mouth every four hours.

My associates and I have for a good many years secured routine blood typing of all patients with involvement of the mastoid, especially children, in whom there seems any possibility of sinus involvement. Thus, we are ready to precede the operation by a supporting blood transfusion and to follow it up by as many others as may be needed. Fluid balance must be maintained by rectal drip or by the intravenous administration of dextrose, but we prefer to save the veins for transfusion. In children, there is no special reason to crowd transfusions beyond 150 cc. each of whole citrated blood; nor should such a quantity be given oftener than each third or fourth day unless the hemoglobin index has fallen below 60. More or less rise of temperature follows each transfusion. We have not had any trouble from retained septic material when the sinus has been smoothly uncovered and either squeezed shut or split open and obliterated by the use of tight rolls of selvedged iodoform gauze packing. These rolls are left in place from five days to a week.

The main point in the treatment of aural disease in cases in which blood cultures are positive or in which one suspects that such cultures may be obtained by proper technic, seems to be swift, shockless and complete operation, preceded or accompanied by blood transfusions.

Blood culture often fails because too little blood is secured and also because it is taken at a period of remission of temperature. We have had better luck with such cultures just before an expected rise in temperature, often at the beginning of a chill. Excellent details respecting methods of culture, particularly as to prognostic value of growth in liquid mediums, have recently been published by Shwartzman and Goldman.[9]

While most otologists have for a good many years employed transfusions as an aid in handling frank sinus thrombosis, it is probable that unavailability of early and accurate typing, as well as of classified donors, still withholds this agency from sufficiently prompt employment in an occasional case. Unless, as in my second case, individual resistance has been carried along by the slow sacrifice of hemoglobin and the leukocytes, metastatic foci will have developed already within the first week and transfusion will be too late. Perhaps, according to Baum's valuable suggestion, immune serums secured from human donors may become available.[10]

It seems surprising that only last year Moritz Weber,[11] of Leipzig, Germany, found it necessary to point out to his elder German colleagues [12] the necessity of frequent transfusions and of registries of donors. Yet, while such things are a commonplace to physicians in the cities, one must not forget that they are out of reach in many communities and that the management of infections of the blood stream associated with mastoiditis must depend on prompt recognition of serious symptoms and transfer of the patient to some institution where every facility is available for reenforcement of the impaired resistance of his circulatory system by repeated transfusions.

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9. Shwartzman, G., and Goldman, J. L.: Streptococcus Hemolyticus Bacteremia, Arch. Surg. **34**:82 (Jan.) 1937.

10. Baum, H. L.: A Method of Specific Treatment in Certain Streptococcus Infections, Arch. Otolaryng. **20**:504 (Oct.) 1934. Hofer, in discussion on Die otogene Sepsis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **38**:108, 1935.

11. Weber, M.: Blutübertragung bei otogener Allgemeininfektion, Ztschr. f. Hals-, Nasen- u. Ohrenh. **37**:425, 1935.

12. (*a*) Uffenorde, W.: Die auf den Bulbus der V. jugul. int. beschränkte Thrombose, Ztschr. f. Hals-, Nasen- u. Ohrenh. **34**:280, 1933. (*b*) Haymann, L.: Ueber Vorkommen und Behandlung otogener Allgemeininfektionen ohne Sinusthrombose bzw. ohne erkennbare Thrombenbildung bei explorativen Massnahmen am Sinus, ibid. **36**:350, 1934. (*c*) Seiferth: Ueber otogene septische Allgemeininfektion unter besonderer Berücksichtigung des Blutbildes und der Lumbalpunktion, ibid. **38**:65, 1935. (*d*) Marx, H.: Die otogene Sepsis, ibid. **38**:3, 1935.

GRAFTS IN THE ROUND WINDOW IN THE TREATMENT OF CERTAIN TYPES OF DEAFNESS

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In 1931 the results of acute experimental fixation of the membrane of the round window in animals were reported,[1] and the following year a further report was made of the effect of placing tissue grafts in the niche of the round window.[2] In the later experiments the animals were kept alive until the grafts had been in place sufficiently long to take, and the hearing was then tested. In many instances, in which the graft was proved microscopically to have actually grown in its new position, there was a striking increase in the functional ability of the treated ear over that of the normal ear. Such measurements were made by estimation of changes in electrical potential in the auditory nerve after acoustic stimulation of the animal's ear. The technical details of the method will be found in the articles mentioned.

These observations have been confirmed repeatedly, and the phenomenon, whatever the explanation may be, is an established one under controlled experimental conditions. The original explanation of the effect is suggested by the title of the article, "Immobilization of the Round Window Membrane." If the membrane, owing to its structure and mobility, seems to damp in some way the sound waves in the intralabyrinthine fluid, the window itself may be functioning normally in the capacity of a safety valve. This conception was further supported in studies on auditory fatigue, in which it was demonstrated that ears with a fixed membrane in the round window become fatigued much more rapidly and to a greater extent than normal ears. Other explanations were based on the derangement of the normal difference in phase between the oval and the round window. It had been shown that the electrical responses at these two windows are of opposite phase under normal circumstances. The rôle of the round window as a safety valve still seems to be the most satisfactory explanation of its function as a factor in actual hearing.

From the Otologic Research Laboratory, Abington Memorial Hospital.

1. Hughson, Walter, and Crowe, S. J.: Function of the Round Window: An Experimental Study, J. A. M. A. **96**:2027 (June 13) 1931.

2. Hughson, Walter, and Crowe, S. J.: Immobilization of the Round Window Membrane: A Further Experimental Study, Ann. Otol., Rhin. & Laryng. **41**:2 (June) 1932.

The possibility of applying this procedure to clinical otology immediately presented itself. Many practicing otologists have noted the effect of placing cotton in the round window of a patient with a large chronic perforation of the tympanic membrane. The patient may experience an immediate subjective improvement in hearing. In some instances he may even learn to put the cotton in place without help. In 1931 Guild [3] drew attention to the striking difference in the extent of loss of hearing in persons with long-standing chronic otitis media dependent on the degree of involvement of the round window with scar tissue. In persons with extensive involvement of the conductive mechanism hearing was affected less if the niche of the round window was filled with scar tissue. Improvement in hearing has also been noted after the introduction of various antiseptic powders into the middle ear, doubtless due to filling of the niche of the round window with the powder.

In November 1931, an attempt was made at the Johns Hopkins Hospital to place a graft of fascia in the round window of a deafened patient. Technical difficulties were encountered, and the operation was unsuccessful. Other operations were performed, the approach being made through a simple mastoid exposure, but again improvement failed to develop and the operation was abandoned.

Were it possible to approach the middle ear through the external canal and the drum and to obtain a reasonably good exposure of the niche of the round window, an important objection to surgical intervention in the treatment of deafness would be overcome. Deafness alone does not warrant major surgical operation as a therapeutic measure. However distressing the condition may be, its correction must not involve risk of life or more than an outside chance of further impairment. The recent rapid improvement in hearing aids places a grave responsibility on the surgeon suggesting radical therapy.

With this fact uppermost in mind, routine otoscopic examinations were made with a binocular head magnifier equipped with a brilliant illuminating system. By actual measurement, such an instrument gives binocular vision at a distance well within the cavity of the middle ear and permits, with its 5 diameters of magnification, a working distance of 10 inches (25 cm.) between the lens and the object.

The selection of patients for possible operation was given most careful consideration. Before operation could be suggested, the following standards had to be met:

1. The deafness cannot be of the nerve type, as proved by adequate bone conduction audiograms.

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3. Guild, Stacy R.: Correlations of Histologic Observations and the Acuity of Hearing, Acta oto-laryng. **17**:2, 1932.

2. There must not be any active infection in the ear, nose or throat. The drum must be intact and reasonably free from scarring, adhesions etc.

3. All other methods of treatment for deafness must have been employed and found ineffective.

4. The loss of hearing must not be more than 50 decibels for the critical frequencies.

5. Age is no limitation provided the deafness is not of the old age or the nerve type.

6. Only one ear, and that the poorer of the two, should be selected for the first operation.

A patient was selected whose condition thoroughly fulfilled these standards, and an operation was performed in the Abington Memorial Hospital on Aug. 26, 1936. Previous experience had emphasized the difficulties arising from hemorrhage of the canal wall caused by too vigorous cleansing, the necessity for a few special instruments and an adequate and reliable suction apparatus. The operative preparation and technic arranged beforehand were used in this and in later cases, with only slight modification.

As general anesthesia is necessary, the patient must be admitted to the hospital on the afternoon before operation. At this time the canal is carefully cleaned, vibrissae removed and the wall washed gently with alcohol and ether. Tribromethanol in amylene hydrate is the anesthetic of choice, with supplementation by ether when necessary. Local preparation in the ward is that usually used for an operation on the mastoid. The patient is placed on the operating table in the prone position, with the shoulders and chest slightly elevated, an important technical point in preventing any accumulation of blood or serum in the cavity of the middle ear. The pinna and the mastoid region are then scrupulously cleaned with alcohol, and the canal is again cleaned gently with alcohol and ether.

An incision from ½ to ¾ inch (1.25 to 2 cm.) long is made at the base of and directly behind the pinna, and a small piece of fascia is removed to be used as the graft. This is kept until required in a sponge saturated with a warm solution of sodium chloride. An ordinary black metal ear speculum equipped with a handle is used for the operative approach.

A broad crescentic incision is made in the posterior half of the drum. It makes no difference whether the base of this flap is toward the annulus or the malleus. Immediately after the incision is made, suction is applied to the free margin of the drum, and the first brisk hemorrhage promptly stops. The loose flap of the drum is then turned out of the way by gentle manipulation with a pledget of cotton. Through this opening some part of the niche of the round window can always be seen, though the entire round window seldom presents directly outward and does so much less in older than in younger persons. The portion of the middle ear thus available for inspection is examined, particularly the promontory, and the margin of the niche is wiped carefully with a pledget of dry cotton. When the normally pale mucous membrane becomes slightly hyperemic, the fascia is placed in the niche of the round window. The fascia graft should

be of sufficient bulk to extend well outside the niche and should be packed firmly in place with a moist pledget of cotton. Care must be taken in this manipulation, as rupture of the delicate membrane of the round window would have disastrous results. Any free blood or serum is removed from the middle ear by suction; the drum flap is turned back into position; a cotton pledget is packed loosely in the canal, and the incision in the skin is dressed. The patient leaves the hospital on the second day after operation and is examined every third day until the drum is healed. An audiogram is made on the day of discharge from the hospital and at each visit thereafter.

Eight operations of this type have been performed. Only the uniformity of the postoperative course warrants report of so small a series.

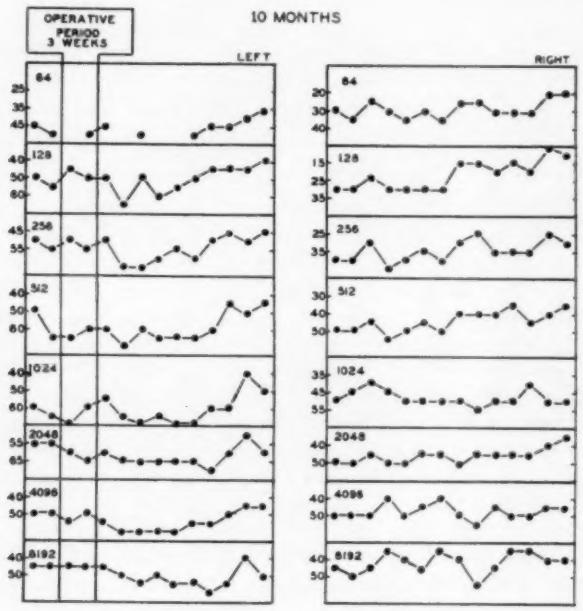

Chart 1 (case 1).—Hearing thresholds before and for ten months after operation.

Brief case histories and composite audiograms for six of these cases are given. In the audiograms each frequency is charted separately, the individual points representing the total number of audiograms made. The frequency is indicated in each separate block, and the figures to the left of each chart represent the loss of hearing below normal in decibels. The caption "operative period" is imposed for the side operated on and represents the time between operation and complete healing of the incision in the drum. The total length of time between the operation and the last examination is indicated.

Case 1.—A girl aged 16 had been deaf since she was 4 years old. She had had scarlet fever at 8 years and tonsillectomy at 10 years. Impairment of hear-

ing was steadily progressive; the usual treatments were of no benefit, and difficulty was encountered in school. Steady improvement was noted in the hearing for the five lower frequencies and for 4,096 double vibrations beginning one month after operation.

CASE 2.—A young woman aged 20 had had a tonsillectomy at 14 years, followed by earache and deafness. Constant treatment produced no improvement, but a gradual increase in deafness occurred. Since closure of the drum gradual improvement has been noted for all frequencies except 4,096 double vibrations. Frequency 8,192 was heard for the first time. At the time of writing she shows marked subjective improvement and can hear sounds which she has never heard before.

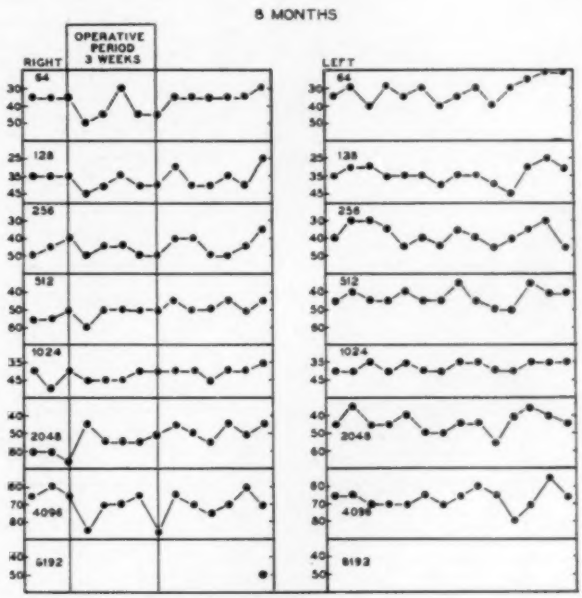

Chart 2 (case 2).—Hearing thresholds before and for eight months after operation.

CASE 4.—A woman aged 35, whose mother and three sisters were deaf, had been deaf since an attack of measles in childhood; tonsillectomy had been performed, and every possible treatment had been given. Chart 3 shows the profound degree of impairment. She was given no encouragement but was anxious to be operated on. She could hear only with a hearing aid. The drum did not heal for three weeks, and during the operative period she had further impairment. Since closure of the drum there has been slight but consistent improvement in acuity of hearing for the critical frequencies. The ear operated on is the better of the two at the time of writing, and she uses the hearing aid on the right instead of on the left side, as she did formerly.

CASE 5.—A woman aged 24 had had abscesses of both ears at 10 years. Deafness was progressive and marked for six years, and the usual treatment was given. In this case a six month interval elapsed between the first examination and the time of operation. Note that impairment increased in this period an approxi-

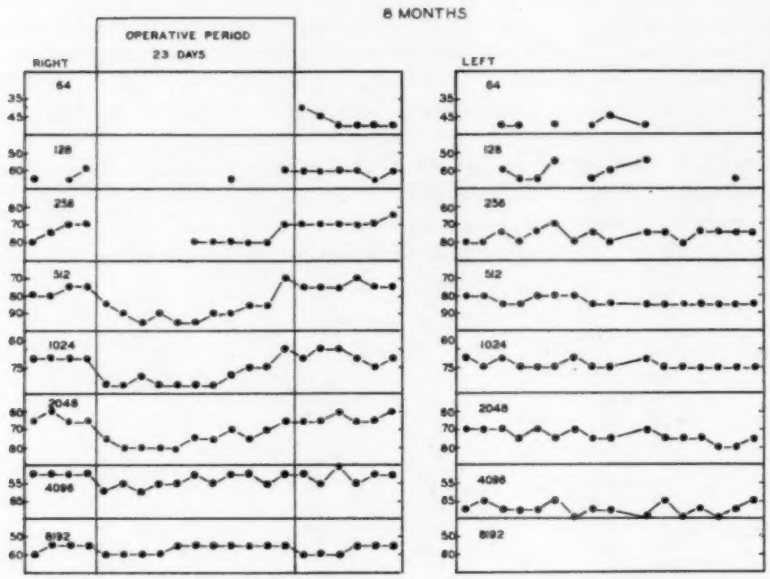

Chart 3 (case 4).—Hearing thresholds before and for eight months after operation.

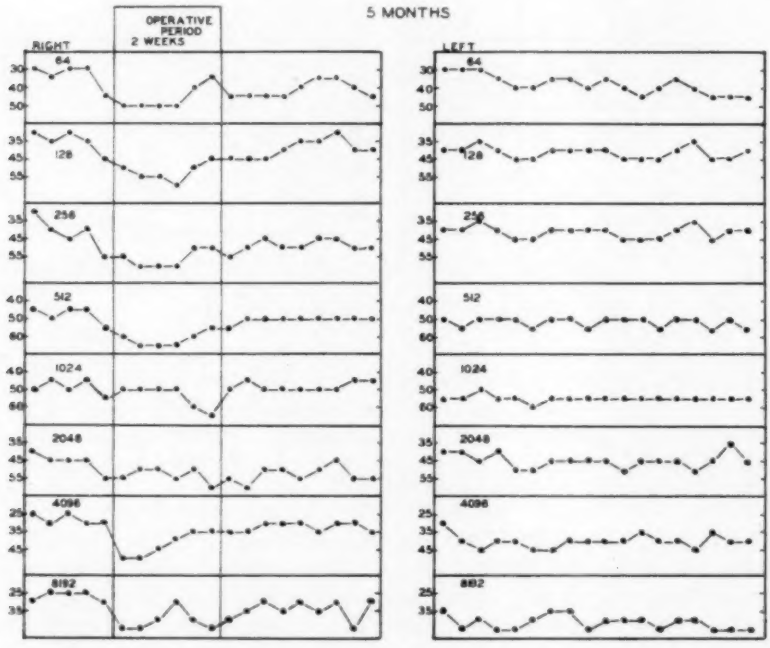

Chart 4 (case 5).—Hearing thresholds before and for five months after operation.

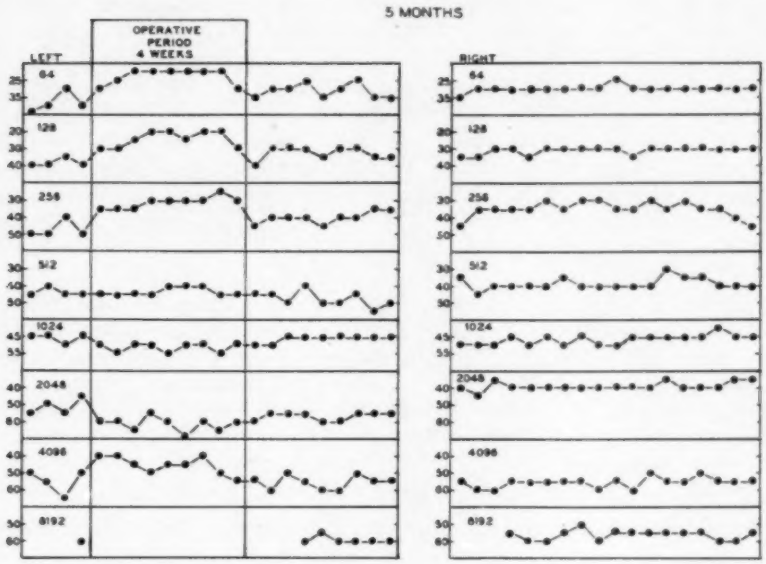

Chart 5 (case 6).—Hearing thresholds before and for five months after operation.

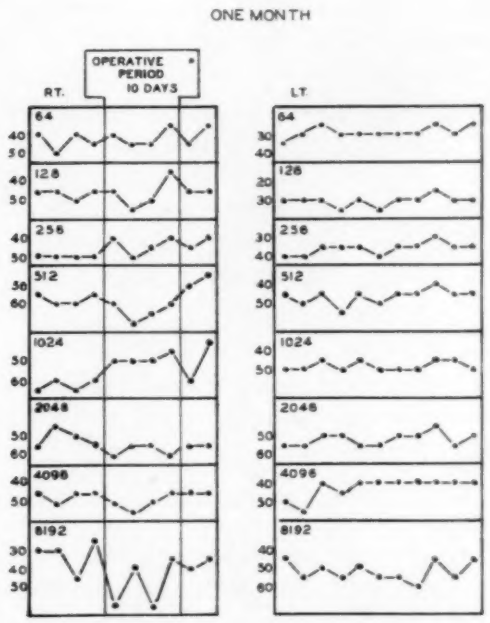

Chart 6 (case 8).—Hearing thresholds before and for one month after operation.

mate average of 15 decibels for the five lower frequencies. Since the drum healed, there has been slow but constant improvement above the immediate preoperative level. Compare the chart with that for the side not operated on.

CASE 6.—A woman aged 45 had been deaf since the age of 11 years. She presented a history of tonsillectomy and intractable facial neuralgia and excessive use of acetylsalicylic acid for many years; every possible form of treatment had been used. Note the marked improvement during the operative period as shown in chart 5. Hearing for the three lower frequencies shows improvement, and 8,192 double vibrations is consistently heard at the time of writing. The hearing in the opposite ear is unusually consistent for all frequencies and has remained relatively unchanged.

CASE 8.—A woman aged 45 had been deaf since childhood; constant treatment was given with no improvement, and the deafness had been rapidly progressive recently. During a preoperative period of six months, weekly dilations and insufflations of the eustachian tube were carried out. She shows a striking subjective improvement and a general audiometric improvement of approximately 15 decibels for the five lower frequencies.

COMMENT

Eight operations for the grafting of fascia in the round window have been performed on persons with a loss of hearing of from 40 to 50 decibels for all the critical frequencies. The cases of six of the patients are reported in detail. The remaining two after an interval of five and three months, respectively, showed a slow but consistent improvement in the ear operated on. In an analysis of the postoperative course in these cases, the following facts are worthy of mention:

1. The graft has remained in position in every case. Direct observation through the opening in the drum during the period of healing has demonstrated this fact. It is reasonable to assume that this fixation represents an actual growth of the graft in position.

2. In no case has there been postoperative infection.

3. The drum has healed with little residual scarring in from ten days to three weeks in every instance.

4. Impairment below the original preoperative level has not occurred.

5. There has been a slight but consistent improvement in the acuity of hearing for certain frequencies in every case, the longest period of observation being ten months.

In view of these rather striking facts it would seem reasonable in the future to select the better ear for the first operation. In the present group of cases the practical success of the procedure has been handicapped by the necessity of considerable improvement before the patient's total hearing could be benefited. Had the better ear been selected originally, the improvement would have been immediately apparent to the patient. Subjective improvement, though present in five cases, is

too unreliable to be regarded as an actual measure of success. Operation on the other ear is now being planned for the two patients operated on first.

The marked impairment present in these cases has also taxed the beneficial effect of the operation rather unreasonably. It is hoped that patients with a less profound loss of hearing may prove appropriate for operation and that the small percentage now regarded as suitable may be gradually increased.

The interesting late improvement can be explained only on the basis of gradual contraction of the graft, with a consequent increase of the effect of immobilization.

CONCLUSION

Eight cases of clinical deafness in which an operation designed to immobilize the membrane of the round window was performed are reported. These patients have all shown improvement, as measured by examination of the audiometric threshold after intervals of from one to ten months after operation.

OTITIC HYDROCEPHALUS

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I. THE LITERATURE

HISTORICAL SURVEY

Oppenheim,[1] in 1899, reported a case of acute nonpurulent encephalitis associated with an acute exacerbation of bilateral chronic otitis media in which the patient recovered without surgical intervention. Oppenheim mentioned that previously he had reported five other cases in which the patient died after unsuccessful exploration for abscess of the brain. He expressed the belief that in cases of this type recovery tends to occur without operation, although in the case presented there were paresis of part of the facial nerve and paresis and paresthesia of the right hand six months after recovery. He expressed the belief that these cases fall into a typical group, in which the characteristics of the condition are motor aphasia and monoplegia faciobrachialis dextra. Oppenheim observed encephalitic scarring in the posterior part of the third left frontal convolution and in the foot of the anterior central convolution in the cases in which necropsy was performed.

In 1902 F. Voss[2] reported three cases in which he believed the condition to be nonsuppurative encephalitis; in one of these cases the disease followed injury to the head. In addition to these three cases, he reported two others which he regarded as instances of similar involvement but in which abscess of the brain was present. Both Oppenheim and Voss apparently believed that a condition such as that observed in the first three cases is a stage in the development of true abscess of the brain but that the encephalitis has failed to progress to suppuration. It is extremely difficult to decide from the evidence presented whether or not the condition in any of these cases should be included in the classification of otitic hydrocephalus, but in certain of them the similarity to diseases which now certainly would be placed in the category is marked.

From the Section on Otolaryngology and Rhinology, the Mayo Clinic.

Thesis presented for membership in the American Laryngological, Rhinological and Otological Society.

1. Oppenheim, H.: Zur Encephalitis acuta non purulenta, Berl. klin. Wchnschr. **37**:201-204 (March 5) 1900.

2. Voss, F.: Drei Fälle von Encephalitis im Anschluss an Otitis media, Ztschr. f. Ohrenh. **41**:223-233, 1902.

Passot,[3] in 1913, presented an extensive thesis on the subject of aseptic meningitis, in which he distinguished two clinical pictures. The first picture consists of a simple increase of intracranial pressure, headache, drowsiness and papilledema, with occasional nausea and vomiting. He considered this picture to be more like that of tumor or abscess of the brain than meningitis. This picture he attributed to a collection of fluid in the subarachnoid space. The second picture, which Passot described as characteristic of internal hydrocephalus, includes slight headache and drowsiness, with the subsequent development of hemiparesis or convulsive seizures of cortical type, which may be unilateral or bilateral. He expressed the belief that if choked disks and increased pressure of cerebrospinal fluid are present in addition they indicate the coexistence of the two conditions. He pointed out that if the condition of increased intracranial pressure is not relieved atrophy of the optic nerve and blindness may develop. Passot advised lumbar puncture for collection of fluid under pressure in the subarachnoid space. For the treatment of hydrocephalus he advised ventricular drainage either by way of the auricular wound or through a clean field of operation in the subtemporal region. If operation on one side fails to relieve the symptoms, he advised drainage of the other ventricle. Passot expressed the belief that the hydrocephalus is attributable to adhesions blocking the foramen of Monro. He failed to establish in what manner his therapy would abolish inflammatory adhesions blocking the foramina, and in this point he may have been at fault. As his therapy was successful in relieving the condition, his paper established admirably the symptoms which are considered characteristic of otitic hydrocephalus and was the first in which a rational hypothesis of causation was set forth and a method of adequate treatment suggested.

Borries,[4] in 1921, presented the thesis that otogenic encephalitis is a pathologically different process from that form of encephalitis which leads to the formation of abscess. He observed in the cases of true otogenic encephalitis a perivascular leukocytic infiltration, with hemorrhage and degeneration of the nervous elements. He did not make it clear, however, why these findings should distinguish the syndrome he described from that of encephalitis progressing toward abscess. Borries recounted two cases of nonsuppurative encephalitis in which there was associated mastoiditis. In the first, the eyes tended to deviate to the right, although there was no true nystagmus. There was a positive Babinski sign on the left but no Kernig sign or stiffness of the neck. The reaction to caloric stimulation was the same in both ears.

3. Passot, Raymond: Méningites et états méningés aseptiques d'origine otique, Thèse de Paris, no. 247, Paris, G. Steinheil, 1913.

4. Borries, G. V. T.: Otogenous Encephalitis, Ztschr. f. d. ges. Neurol. u. Psychiat. **70**:93-101, 1921.

On lumbar puncture, the spinal fluid was found to be bloody; 65 per cent of the cells were leukocytes and 35 per cent lymphocytes. Smear and culture of the spinal fluid gave negative results. Paralysis of the left side of the face finally appeared. The right temporal lobe and the cerebellum were explored with negative results, and the patient gradually recovered. In the second case there were symptoms of intracranial involvement in association with labyrinthitis and osteomyelitis following bilateral mastoidectomy. Exploration of the brain gave negative results, and on lumbar puncture the fluid was sterile. Borries expressed the opinion that it is impossible to make a differential diagnosis between abscess of the brain and nonsuppurative encephalitis and that incision of the dura seems to have a favorable effect in encephalitis. He expressed the belief that the principal lesson to be learned from the study of these cases is that when exploration of the brain gives negative results the surgeon should await events rather than explore the brain again. It would seem that the condition in the second case of Borries more nearly corresponds to the symptom complex of otitic hydrocephalus.

Holger Mygind,[5] in 1922, in reporting thirty-eight cases of benign purulent meningitis, included seven in which "optic neuritis" was present. In one case, in which an abscess of the brain was suspected on account of the presence of deep stupor and slow pulse, the dura was opened, the brain explored and a piece removed for microscopic examination; this revealed evidence of considerable acute leptomeningitis (but not the expected encephalitis), with fibrinous exudate on the surface. Of fifteen cases of "collateral meningitis" in which the cerebrospinal fluid was clear, there were several in which some of the symptoms evidently suggested abscess of the brain. Some of Mygind's patients evidently could be considered to have otitic hydrocephalus, but the evidence is completely clear in respect to only one.

Adson [6] recognized the existence of a clinical entity which he termed "pseudobrain abscess." He reported three cases in which there was associated mastoiditis; choked disks and other neurologic signs which indicated the presence of an abscess of the brain. In one of these cases the cerebrospinal fluid was under increased pressure but the cell count and other examinations of the spinal fluid gave negative results. In two exploration for abscess of the brain was made, but no abscess was found. At operation an accumulation of fluid in the subarachnoid spaces and congestion of the cerebral convolutions were noted.

5. Mygind, Holger: Benign Forms of Otogenic Meningitis, J. Laryng. & Otol. **37**:597-612 (Dec.) 1922.

6. Adson, A. W.: Pseudobrain Abscess, S. Clin. North America **4**:503-512 (April) 1924.

Adson expressed the opinion that the symptoms were attributable to localized encephalitis, and he regarded this as a reason for delaying exploration in cases of suspected abscess of the brain, for the condition may undergo spontaneous resolution. He advised exploration in the quiescent stage and expressed the belief that decompression may be of value in relieving the pressure over a region of localized encephalitis. Cases of the sort Adson described are probably, however, of the group in which the condition is now classified as otitic hydrocephalus.

Yerger,[7] in 1925, reported three cases of "acute toxic meningo-encephalitis" in association with mastoiditis and one case in which the condition was in association with acute frontal sinusitis. In three of these cases the symptoms suggested cerebrospinal meningitis, but, according to Yerger, studies of the spinal fluid established the diagnosis of meningo-encephalitis, the patients recovering after drainage of the exciting focus of infection. One patient gave signs suggestive of cerebellar abscess, and exploration was made, the results were negative, and the patient recovered. Yerger expressed the belief that toxins from disease produce serous or sympathetic meningitis; however, he thought that not all the symptoms were the result of irritation of the meninges by the toxins of the disease but that irritation of the whole brain substance was present also.

Symonds,[8] in 1927, reported three cases which were very similar to Yerger's cases and in which a diagnosis of abscess of the brain was made. In two cases the brain was explored; one of the patients refused to submit to operation. In one case there was choking of the disks, with some alteration of the reflexes on the side of the body opposite to that on which the lesion was supposed to be; the findings suggested a lesion of the temporal lobe. In the second case there were headache, drowsiness and right homonymous hemianopia, which suggested a lesion of the left temporal lobe. In the third case, about a month after mastoidectomy on the right side, nausea and vomiting appeared, together with an extensor plantar response on the left, with diminished abdominal reflexes on the same side. Lumbar puncture at the time of operation gave a clear fluid, apparently under increased pressure but not containing an increased number of cells or an increased concentration of protein. The dura mater was opened, and the brain was explored, but an abscess was not discovered. The patient began to improve in about two weeks and eventually made almost complete recovery. Symonds expressed the belief that a "presuppurative" stage of abscess of the

7. Yerger, C. F.: Acute Toxic Meningo-Encephalitis of Otorhinogenic Origin, Arch. Otolaryng. **1**:198-208 (Feb.) 1925.

8. Symonds, C. P.: Some Points in the Diagnosis and Localization of Brain Abscess, J. Laryng. & Otol. **42**:440-448 (July) 1927.

brain was present in these cases in which there were engorgement, exudation and swelling without necrosis. He expressed the belief that in the case of pseudo-abscess the inflammatory process becomes arrested at this stage and resolution then occurs without formation of pus, but in a later paper [9] he altered his conception of the pathologic picture to agree with that of Passot. He expressed the belief that the point of importance to be gained from these observations is that when the general and localizing signs of cerebral abscess are present a negative result from exploration does not necessarily mean that an abscess has been missed. In such a case it might be wise to await the possibility of spontaneous cure.

Cairns,[10] in 1930, in discussing the differential diagnosis of abscess of the brain, said:

> The second condition that I wish to describe more fully is that known as arachnoiditis or serous meningitis. This condition simulates cerebellar abscess rather than temporosphenoidal abscess. It comes on after acute mastoiditis and chronic mastoiditis especially when the labyrinth has been infected. The main clinical feature is papilledema, often of extreme degree, and the headache and other general symptoms may be so slight that the condition is not recognized until sight is seriously impaired.

Cairns reported the case of a boy aged 5 years, who had headache and vomiting immediately after tonsillectomy that was performed five months after mastoidectomy. The temporal lobe was explored with negative results. Two months later the boy's mother noticed that he tripped over curbstones. A month and a half later examination of the disks disclosed severe papilledema and atrophy, and there was exudate around each macular region. To quote again: "Lumbar puncture pressure 230 m.m." There was no rise of pressure when the left jugular vein was compressed. The cerebrospinal fluid was clear. It contained 1 cell per cubic millimeter and 0.02 per cent protein. Cultures were sterile.

In commenting on this case, Cairns stated:

> As far as it is possible to judge from the available evidence this appears to be a case of the so-called arachnoiditis or serous meningitis. The pathology of arachnoiditis is at present quite obscure. In the only case of the condition in which I have been able to make a histologic examination the arachnoidea showed no evidence of inflammation, and I think the term arachnoiditis is probably an incorrect one. All that we know about the pathology of the condition is that there is hydrocephalus, but whether this is due to increased formation of cerebrospinal fluid or to decreased absorption of cerebrospinal fluid, or to a combination of both these factors is at present unknown. The condition follows middle ear and mastoid infection, but it may also occur without any demonstrable focus of

9. Symonds, C. P.: Otitic Hydrocephalus, Brain **54**:55-71 (April) 1931.

10. Cairns, Hugh: Abscess of the Brain, J. Laryng. & Otol. **45**:385-397 (June) 1930.

infection, either in the ears or elsewhere. . . . The case is typical in the absence of severe headaches except for a short period at the beginning of the illness. . . . The condition tends to recover spontaneously. I have seen two cases, both in children, in which perfect recovery occurred without any treatment at all. In one of these the papilledema reached 4 diopters before it began to subside; in the other the swelling was never higher than 2 diopters. In both cases vision eventually became normal. In other cases, however, although all the symptoms clear up, they do not do so until vision is seriously affected. . . . From my experiences with this condition, I believe that a cerebellar decompression, performed before the destruction of the optic discs has become far advanced, is a sure method of preserving vision. It should be carried out in every case in which the papilledema becomes at all severe or in which there is any sign of lowering of visual acuity. . . . The pressing need in this type of case is early diagnosis and as the neurological signs are scanty, this depends on the employment of ophthalmoscopy in all cases of ear disease that show any signs of intracranial disturbance. Even if the headaches and vomiting are severe in the early stages, they are always very much better at the time when the patient is going blind. . . . This condition of "arachnoiditis" comprises a part of that mixed group of cases known as "pseudo-brain abscess."

Key-Åberg,[11] in 1926, reported a case in which paralysis of the left side of the face and paresis of the left arm developed after mastoiditis on the right side. At exploration of the wound in the mastoid the dura was found to bulge. When this was incised, cerebrospinal fluid gushed out under extreme pressure. Lumbar puncture, done a week later, yielded normal spinal fluid. The patient slowly made complete recovery. Key-Åberg expressed the opinion that the difficulties in distinguishing between otogenic encephalitis and abscess of the brain are of no importance, for in all cases of such involvement exploration should be made, not only because of the occasional finding of an extradural abscess but also because opening the dura, even with negative findings, seems to produce a favorable effect. It is possible that Key-Åberg was dealing with a circumscribed, subdural collection of fluid rather than with generalized otitic hydrocephalus.

O. Voss,[12] in 1928, reported two cases in which he termed the condition "acute toxic encephalitis in association with acute, influenzal otitis media" and two cases of encephalitis serosa "without any sign of serous meningitis." He expressed the belief that these findings established the existence of a nonsuppurative type of otogenic encephalitis.

Symonds,[9] in 1931, reported three cases, in one of which operation was performed, with the discovery of internal hydrocephalus; in the two others, the discovery of normal cerebrospinal fluid led to the

11. Key-Åberg, Hans: Contribution à l'étude de l'encéphalite otogène, Acta oto-laryng. **10**:75-80, 1926.

12. Voss, O.: Otitis media und Encephalitis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **21**:596-608 (May 10) 1928.

correct diagnosis. He expressed the opinion that as a not uncommon complication of otitis media there may occur a state of increased intracranial pressure, attributable to the presence of an excess of normal cerebrospinal fluid. He suggested the designation otitic hydrocephalus for this condition, because the term implies no active process of inflammation and, since it is free from the qualification internal or external, it includes conditions in which fluid is within both the ventricles and the subarachnoid space. Symonds expressed the belief that the condition is attributable either to excessive secretion from the choroid plexus or to defective absorption through the arachnoid villi. He stated: "The hypothesis of an obstructive internal hydrocephalus due to inflammatory occlusion of the effluent channel from the ventricles has also to be considered, but the high pressures recorded with the manometer and the large quantities of fluid withdrawn in several cases are against this explanation." He stated that, although in some cases recovery appears to have been spontaneous, the rational line of treatment is drainage of the excessive cerebrospinal fluid by lumbar puncture. If the lumbar puncture fails to relieve the symptoms, Symonds advised that ventricular puncture be undertaken in the first place on the right side, whatever the side of the aural lesion, and if this fails to relieve symptoms that the left ventricle also be tapped. He pointed out the extreme difficulty of distinguishing this condition from abscess of the brain if a preliminary stage of meningitis has caused a temporary excess of cells and protein in the cerebrospinal fluid.

Carmack,[13] in 1933, reported a case in which the condition was similar to that observed in association with otitic disease attributable to suppuration in the sphenoid sinus. He said: "While the cases reported tend to recovery, especially with removal of contiguous infection and spinal drainage, we are impressed with the possibility of these being the result of meningeal irritation from infection with meningitis a potentiality." He expressed the belief that suppuration in any pneumatic space contiguous to the capsule of the brain can produce similar symptoms.

Garland and Seed,[14] in 1933, reported two cases, in one of which definite meningitis, with organisms, increase in number of cells and increase in concentration of protein in the spinal fluid, followed the earlier development of symptoms of "otitic hydrocephalus." The patient made an eventual recovery.

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13. Carmack, J. W.: The Relation of Increased Intracranial Pressure to Disease in the Pneumatic Spaces, Ann. Otol., Rhin. & Laryng. **42**:364-371 (June) 1933.

14. Garland, H. G., and Seed, G. S.: Otitic Hydrocephalus, Lancet **2**:751-753 (Sept. 30) 1933.

Warner,[15] in 1933, reported a case of so-called idiopathic hydrocephalus, in which the condition was successfully treated by repeated puncture of the lateral ventricle. He expressed the opinion that the value of removing as much cerebrospinal fluid as possible in such cases is attributable to the breaking of a vicious circle and the relief of congestion of the choroid plexus. This case suggests that whether hydrocephalus is the result of increased secretion or of decreased absorption, relief of the pressure by ventricular puncture will allow restitution to normal conditions even in so-called idiopathic hydrocephalus.

Smith,[16] in 1934, reported a case in which primary signs suggesting otitic hydrocephalus were followed by definite suppurative meningitis, with organisms present in the fluid. The patient recovered after mastoidectomy.

Nielsen and Courville,[17] in 1934, attempted classification of the cases of "pseudo-abscess" of the brain into five groups, with the first group subdivided into two subgroups. One case was reported to illustrate each of the six types. Group 1 includes cases of disease of the brain without suppuration and without thrombosis of the lateral sinus. The two subgroups of group 1 are as follows: (a) cases in which the patients are adults and intracranial pressure is a prominent symptom and (b) cases in which the patients are young children and present a syndrome which frequently is transient and is characterized by convulsions or paralysis. Group 2 includes "cases associated with sinus phlebitis." Group 3 is composed of "cases of certain lesions of the posterior cranial fossa, as a tumor, basilar meningitis or ependymitis." Group 4 includes "cases of otogenic leptomeningitis," and in group 5 are "miscellaneous cases including multiple emboli, thrombosis of cerebellar artery, syphilis, etc., occurring in the presence of otitis media." Although Nielsen and Courville based their classification on an extensive review of the literature, from which, however, the interesting papers of Passot and of Symonds were omitted, it seems that they could conceive of no integrating pathologic basis for any of their so-called groups, the only thing in common being the difficulty in distinguishing the condition from abscess of the brain. In conclusion, they wrote:

> In view of the difficulties pointed out, how are we then to determine when in the course of otitis media an abscess of the temporal lobe or cerebellum has

15. Warner, E. C.: Hydrocephalus Successfully Treated by Repeated Puncture of the Lateral Ventricles, Proc. Roy. Soc. Med. **26**:1007 (June) 1933.

16. Smith, C. H.: Symposium on Facial Nerves: II. Otitic Hydrocephalus, with Report of a Case, Laryngoscope **44**:931-934 (Dec.) 1934.

17. Nielsen, J. M., and Courville, C. B.: Pseudo-Abscess of the Brain: Intracranial Disease During Otitis Media Simulating Encephalic Abscess, Ann Otol., Rhin. & Laryng. **43**:972-987 (Dec.) 1934.

developed and needs exploration? The answer is—Not by one visit or even one careful examination but by repeated systematic follow-up examinations, plus a knowledge of what is apt to develop in such a case. To this is added diagnostic surgery which we do not hesitate to advise. When the eardrums have been opened but symptoms continue we study the mastoids. When the mastoids have been drained, the dural and sinus plate removed, and the lateral sinus studied, one has determined the presence or absence of extradural abscess and lateral sinus thrombosis. These procedures will, if carefully carried out, not only remove infection and hasten recovery but will place the examiner in a position to know whether extension has occurred. This greatly simplifies the diagnosis. If the patient is known not to have extradural abscess, subdural abscess or meningitis, yet symptoms and signs of intracranial complications are still present, exploration for abscess of the brain is certainly justified.

Rabbiner,[18] in 1935, reported a case of pseudo-abscess of the brain associated with mastoiditis. In this case meningitis sympathica was present together with choking of the disks, which reached from 2 to 3 diopters, and bilateral palsy of the abducens nerve. A diagnosis of abscess of the brain above the tentorium was made, but it was decided to observe the patient, who eventually made complete recovery. The choking of the disks disappeared in about two months.

Hampson,[19] in 1935, advised continuous drainage for cerebrospinal meningitis when a dry tap or other evidence of almost certain fatal termination is present. He reported recovery in six of twenty-five cases.

PATHOLOGY, PHYSIOLOGY, SYMPTOMATOLOGY AND TREATMENT

In writing of abscess of the brain Oppenheim[20] said that otitic cerebral abscess is not always immediately recognized because, especially in the young, a number of its phenomena occur in cases of uncomplicated otitis, particularly when there are exacerbations of the chronic process and retention of pus; to these phenomena belong headache, vomiting, vertigo, stupor, fever and even pupillary differences. Some observations even showed that optic neuritis and ptosis might be attributable to uncomplicated otitis. All these phenomena had been made to disappear, for example, by paracentesis of the tympanic membrane. Whenever these symptoms appear in the course of aural disease, a local cause, such as retention of pus, must be thought of. External purulent pachymeningitis or extradural abscess, which Oppenheim stated is the most frequent complication of aural suppuration, may cause almost

18. Rabbiner, Max: Otitic Meningitis—Pseudo Brain Abscess, Laryngoscope **45**:676-684 (Sept.) 1935.

19. Hampson, A. C.: Cerebrospinal Meningitis with Special Reference to the Treatment of Associated Hydrocephalus, Guy's Hosp. Rep. **85**:431-446 (Oct.) 1935.

20. Oppenheim, H.: Lehrbuch der Nervenkrankheiten, ed. 6, Berlin, S. Karger, 1913, vol. 2, pp. 992-1132, 1137, 1254 and 1261.

all the symptoms of cerebral abscess. Atypical otitic sinus thrombosis, resembling abscess, has been observed. The greatest difficulty lies in distinguishing abscess from diffuse purulent meningitis. In some cases in which the course is atypical, the clinical picture resembles that of abscess. Oppenheim said that serous meningitis, not a rare complication of purulent otitis, may simulate cerebral abscess on the one hand or purulent meningitis on the other but does not, as a rule, cause any focal symptoms (bilateral paresis of the extremities and cranial nerves and cerebellar ataxia have been noticed several times) or any marked or continuous rise in temperature. Choking of the disks and visual disorder are observed early and often, and, what is very important, they may spontaneously disappear or recede after evacuation of the pus from the ear or from the extradural region or after opening of the arachnoid space.

In regard to acute hydrocephalus Oppenheim wrote:

> Quinke thinks that a serous meningitis of the ventricles, a simple serous inflammation of the intracerebral pia, may be the cause of the internal hydrocephalus in not a few cases. The exudate arises chiefly from the choroid plexus, the cortical pia, however, may be the seat of a simple inflammation and produce an external hydrocephalus. Further observations have shown that this form of meningitis is also not rare in purulent otitis. If it develops acutely, it is difficult to distinguish it from purulent meningitis. The rise of temperature is, however, absent, or is only slight and inconstant, or the fever which had been present at the commencement soon recedes. The visual disturbance is very marked in these cases. An absolute diagnosis can generally be made only from the further course as this acute acquired hydrocephalus, or acute serous meningitis ends in complete or partial recovery in some weeks or months. Some cases, however, in which the symptoms (headache, vertigo, vomiting, optic neuritis, cerebellar ataxia) resemble the chronic form, or in which an ocular affection forms the residual symptom of a cerebral disease, become chronic.

In cases of acute involvement which resembles meningitis Oppenheim advised treatment similar to that for meningitis. Direct ventricular puncture is preferred by some surgeons, particularly when spinal puncture is unsuccessful. In some cases division of the dura mater over the brain relieves the symptoms. Recovery is produced especially often in the otitic form of this disease. Von Beck [21] observed the disappearance of coma and Cheyne-Stokes respiration and the return of vision in a few hours from the influence of ventricular puncture.

Bateman [22] wrote that the cerebrospinal fluid, like all other fluids of the body, is formed from the blood. The choroid plexuses, with their secretory cells, have, in a physiologic sense, the same selective functional activity as have the secretory cells of the parotid, lacrimal

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21. von Beck, cited by Oppenheim.[20]

22. Bateman, J. F.: Meningitis with Special Reference to the Rôle of the Pacchionian Bodies, Ohio State M. J. **25**:970-976 (Dec.) 1929.

or pancreatic glands. It is generally believed, and in part substantiated by experimental evidence, that the bulk of the fluid arises from the choroid plexuses of the lateral, third and fourth ventricles. The fluid has a slow but constant circulation. It passes through the entire ventricular system, then upward through the tentorial notch and over the cerebral cortex, where it reaches its final destination, the pacchionian bodies, through which it passes into the blood stream. The pacchionian bodies are tuftlike, spongy masses of arachnoid tissue that do not contain blood vessels, lymphatics or nerves and consist of a dense interlacement of flattened bundles of fine, fibrous tissue interspersed with elastic fibers and platelike cells. These small tufts of arachnoid tissue are covered externally with endothelium. They push before them the greatly attenuated dura, and overlaid by the latter and the endothelial lining of the blood space they project into the superior longitudinal sinus or its lateral diverticula. The pacchionian bodies occur in patches and are situated, for the most part, on each side of and close to the longitudinal dural sinus. Bateman stated that the anatomicopathologic conditions of the pacchionian bodies may be divided into two types, the acute and the chronic. In either condition the ultimate outcome is obstruction, either complete or partial. In the acute inflammatory processes within the meninges, the cellular exudate is most pronounced in and around these clusters of arachnoid granulations. The separate villi become filled with polymorphonuclear leukocytes, and there is a corresponding increase in the mesothelial cells lining the villus. Some of these processes advance to the degree of colliquation and produce complete obstruction directly or indirectly through rupture of the neck of the villus, with escape of the fluid and exudate into the subdural cavity. Bateman expressed the belief that of all cerebral lesions abscess has the most variable course. Some are acute and of short duration; others rapidly become encapsulated and remain for years. In the case of cortical abscess there is usually associated meningitis, either local or diffuse. Extension is always with the course of the cerebrospinal fluid, and the amount of destruction depends solely on how well the exudate and necrotic tissue can be removed. If the only avenue of escape of the fluid becomes occluded, symptoms become markedly exaggerated and death usually ensues before surgical intervention is possible. Bateman further stated that from an anatomic point of view any inflammatory process within the meninges may be considered obstructive. Any form of increased intracranial pressure, including serous meningitis, may be considered obstructive. There is not a single example in normal physiology in which nature provides for overstimulation of secretory cells to the extent that the part becomes pathologic as a result of increased activity. Many cases of so-called serous meningitis are recorded, and the condition is said to be attributable

to oversecretion of the fluid. This in part may be true, but such a view is entirely in opposition to physiologic principles. According to Bateman,[22] the aforementioned histopathologic observations seem to indicate that the pacchionian bodies bear the brunt of any subarachnoid inflammatory process and that lumbar drainage is of no therapeutic value. In considering the effect of stagnation of cerebrospinal fluid occurring in cases in which the foramina of Luschka are closed, Bateman [23] came to the conclusion that inadequate circulation, with stagnation of spinal fluid and accumulation of waste products, would lead to deterioration of nerve substance.

Boschi,[24] in 1933, comprehensively reviewed the literature on the secretion, direction of flow and absorption of the cerebrospinal fluid. He stated that in some cases of symptomatic hydrocephalus no abnormality has been found but edema of the subependymal tissue. But in other cases desquamation of the ependyma has been found, with proliferation of the subependymal glial cells. In the choroid plexus one finds sometimes a completely normal appearance; at other times the plexus is hypertrophied, there is marked degeneration of the epithelium and cellular infiltration of the perivascular tissues is evident. The most profound alterations of the plexus are observed in meningitis. However, Boschi remarked that the presence of ventricular hypertension remains inexplicable in cases in which the choroid plexus is normal if one wishes to attribute the condition exclusively to pathologic change in the plexus. Boschi pointed out that, in addition to means of escape through the pacchionian bodies and arachnoid villi, pathways of escape exist along the cranial and spinal nerves, which are considered by many observers to be the routes of major importance.

Bedford,[25] in 1934, was unable to produce hydrocephalus in dogs by occluding the vein of Galen and was unable to confirm the work of Dandy and Blackfan.[26] He concluded that in the dog hydrocephalus does not follow experimental occlusion of the great vein of Galen. He suggested that the hydrocephalus which was observed occasionally by Dandy and Blackfan in dogs in which the great vein of Galen had been occluded is not attributable to obstruction of this vessel but is of an

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23. Bateman, J. F.: Closed Foramina of Luschka in the Brains of the Insane: Their Influence on Pathogenesis of Psychoses, Arch. Neurol. & Psychiat. **14**:616-637 (Nov.) 1925.

24. Boschi, Gaetane: Méningites sérouses internes ou ventriculaires, Rev. neurol. **1**:851-878 (June) 1933.

25. Bedford, T. H. B.: The Great Vein of Galen and the Syndrome of Increased Intracranial Pressure, Brain **57**:1-24 (March) 1934.

26. Dandy, W. E., and Blackfan, K. D.: Internal Hydrocephalus: An Experimental, Clinical and Pathological Study, Am. J. Dis. Child. **8**:406-482 (Dec.) 1914.

entirely different etiology. Bedford quoted Cushing to the effect that the results obtained by Dandy and Blackfan probably are attributable to blocking of the arachnoid villi by blood and the products of inflammation.

Barré,[27] in the same year, expressed the belief that there is definite correspondence between spinal arachnoiditis, arachnoiditis of the posterior fossa, the lesions of the angle described by Eagleton [28] and the arachnoiditis frequently observed in otologic services.

Fanconi,[29] in 1934, divided hydrocephalus following infection into that attributable to hypo-absorption and that attributable to hypersecretion.

II. COMMENT ON THE LITERATURE

PATHOLOGY AND SYMPTOMATOLOGY

It is evident that hydrocephalus may accompany any inflammatory condition in which toxins or bacteria reach the arachnoid in sufficient concentration to produce inflammatory change. Thus are included many generalized, acute inflammatory conditions, and the neurologic signs which accompany acute infections in childhood suggest that in children toxic products more easily reach the meninges. The preponderance of cases of acute hydrocephalus in association with otitic disease is in all probability attributable to the frequency with which inflammation of the dura is associated with this condition. Otitic hydrocephalus used as a descriptive term for this condition is perhaps at fault in unmistakably implying association of this condition with disease involving the temporal bone.

The material secured in cases of otitic hydrocephalus has been meager and has shown little pathologic change. Encephalitic scarring was noted by Oppenheim, but inflammatory changes in the arachnoid of moderate degree, without evidence of encephalitis, have usually been observed. Bateman's evidence seems to locate the lesion, in the majority of cases, in the arachnoid villi and the pacchionian bodies, with blocking of the foramina of Magendie and of Monro relegated to a secondary place. Other observers have expressed the belief that the principal site of absorption is along the arachnoid prolongation accompanying the cranial and spinal nerves. The hypothesis that hydrocephalus is produced by increased secretion, possibly attributable to interference with flow through the great vein of Galen, seems to have

27. Barré, J. A.: Étude sur l'arachnoidite spinale et l'arachnoidite de la fosse cérébrale postérieure, Rev. neurol. **1**:879-918 (June) 1933.

28. Eagleton, W. P.: Localized Bulbar Cisterna (Pontile) Meningitis, Facial Pain and Sixth Nerve Paralysis and Their Relation to Caries of the Petrous Apex, Arch. Surg. **20**:386-420 (March) 1930.

29. Fanconi, G.: Zur Diagnose und Therapie hydrocephalischer und verwandter Zustände, Schweiz. med. Wchnschr. **64**:214-223 (March 10) 1934.

little support, judging from the experimental and pathologic evidence submitted. It would seem that the hypothesis that hydrocephalus is produced by interference with the mechanism of absorption is supported by the great weight of evidence. Since this lesion may follow any inflammatory or obstructive lesion of the pacchionian bodies or arachnoid prolongations along the nerves, it must follow that hydrocephalus often means the presence of serious inflammation involving the dura and that either presence or absence of cells in the cerebrospinal fluid is not of major diagnostic importance. It is possible that in some stage of the development of acute hydrocephalus an increased cellular content of the cerebrospinal fluid will be a constant finding and that in some cases, as the inflammation recedes, the pressure of the hydrocephalus on the cranial sinuses, especially in the region of their exits through the cranium, is sufficient to establish a vicious circle and cause a continuation of the hydrocephalus by mechanical means. The appearance of true suppurative meningitis following so-called otitic hydrocephalus, without an increased number of cells in the cerebrospinal fluid, would seem to cast some doubt on the supposition that so-called serous meningitis has a benign prognostic import and that only the so-called meningitis sympathica suggests the presence of serious disease in the vicinity of the dura. It is hardly possible that any intradural inflammation could be uncomplicated by the presence of acute hydrocephalus in some degree, and it is therefore not strange that some difficulty is experienced in distinguishing between other types of intradural inflammation and acute hydrocephalus. In cases in which on histologic study the only apparent lesion is in the pacchionian bodies, the most frequent sign produced is papilledema, but projectile vomiting, slow pulse, drowsiness, stupor, aphasia, paresis and incoordination, sometimes simulating perfectly the symptoms and signs of cerebral or cerebellar lesion, even to cytologic and chemical changes in the cerebrospinal fluid, may be present. The greater difficulty, therefore, would seem to be in deciding, in a case of encephalitis, which symptoms are produced by involvement of the brain tissue and which by the almost inevitably present hydrocephalus. It would perhaps be an exaggeration to say that the general symptoms of encephalitis are those produced by the accompanying hydrocephalus, but it would probably be nearer to the truth than to ignore the fact of hydrocephalus and to attribute all the symptoms to encephalitis or to edema of the brain.

It is possible that in certain instances a minimal degree of encephalitis has been present in the type of case under consideration, but the characteristic symptoms have been observed in its absence, and the effect of the encephalitis, if appreciable, has apparently been slight.

It would seem in considering the literature that in certain cases acute hydrocephalus not associated with any intradural lesion simulates

encephalitis or abscess of the brain so closely that the diagnosis can be made with difficulty or not at all. For this condition the secondary classification of pseudo-abscess, suggested by Adson, would seem properly to designate a distinct clinical type, and for that reason should be retained. It is in cases of this condition and in the group comprising cases of threatened atrophy of the optic nerve that it is most important to come to a decision concerning the proper therapeutic measures.

TREATMENT

It is apparent that in most cases of acute hydrocephalus there is a favorable response to elimination of the exciting focus, with drainage of large quantities of cerebrospinal fluid by lumbar puncture. Some objections to spinal drainage have been raised on the ground that localized meningitis may thus become generalized. However, the experience of most otolaryngologists seems to have been that if the exciting focus has been eliminated, this generalization of the infection does not occur. Bateman, however, has insisted that this is a real danger and that spinal or cisternal drainage in cases of meningitis, when organisms are present in the spinal fluid, is illogical. It is my opinion that this danger can be overemphasized and that in most cases of acute otitic hydrocephalus, spinal drainage, after the exciting focus has been eliminated, is the procedure of choice. However, in many cases in which choked disk is a prominent symptom, it is seen from reports in the literature that from one to two months has passed before the papilledema has disappeared and in some instances secondary atrophy of the optic nerve has taken place, with partial or complete blindness. Oppenheim, in 1900, reported the rapid disappearance of choking of the disks after ventricular puncture, and this result has been obtained recently in several instances. When papilledema is a prominent symptom and fails to recede under spinal drainage, especially when there is any failing of vision, the rapid relief afforded by ventricular puncture would seem to make it the procedure of choice.

It is also probably true that when other symptoms of hydrocephalus fail to respond to spinal drainage ventricular puncture is to be preferred to prolonged illness. When the type of acute hydrocephalus known as pseudo-abscess is present, especially when differential diagnosis is made impossible by increase in the cell content of the cerebrospinal fluid, the advantages of ventricular puncture would seem to be obvious. In the presence of encephalitis, rapidly advancing stupor may be attributable to the effects of acute hydrocephalus, and ventricular puncture may enable the patient to survive until localization has taken place and optimal conditions for surgical drainage of purulent material have become established.

III. ILLUSTRATIVE CASES

Four cases are presented; three are instances of pseudo-abscess of the brain and one of localized purulent meningitis associated with subacute internal hydrocephalus. These cases illustrate some of the points considered.

CASE 1.—A girl aged 10 years was presented for examination with the history that after an acute infection of the upper part of the respiratory tract, accompanied by cough and sore throat, bilateral earache developed. The ear drums ruptured spontaneously, and large quantities of serosanguineous fluid were discharged. A week later high fever developed, and three days before her admission to the clinic she had a chill followed by a rise in temperature to 103 F.

On examination a profuse discharge from the right auditory canal and some concentric narrowing of the external auditory canal were noted. Tenderness over the mastoid was absent. The remainder of the examination of the ear, nose and throat revealed nothing of significance. Examination of the eyes revealed normal reflexes, ocular motion and fields. There was early choking of the disks—1 to 2 diopters on the right and 2 diopters on the left. Neurologic examination gave negative results, except that the plantar response on the right was extensor. The spinal fluid was clear and under moderately increased pressure. Smears and cultures revealed no organisms. Lymphocytes numbered 3 per cubic millimeter, and the concentration of protein was 30 mg. per hundred cubic centimeters. A roentgenogram of the mastoids gave evidence of mastoiditis on the right. A blood culture, made on the day of admission, contained no growth in forty-eight hours. General physical examination revealed no pertinent findings. The child was kept under observation, and on the third day became very drowsy. It was thought advisable to clear up the exciting focus in the mastoid and to observe the course of events.

At operation no evidence of breaking down of the mastoid cells was found, nor was any pus discovered except in the antrum. The bone bled freely. The tegmen was removed, and the dura of the temporal lobe appeared slightly reddened. The sigmoid sinus was uncovered from the upper to the lower turn. There was no reddening of the dura, and the sinus filled readily from above and below. The immediate postoperative condition was good, but on the fourth day after operation projectile vomiting developed. Examination at that time disclosed bilateral choking of the disks of 3 diopters. On the fifth day the pack was removed from the mastoid; this produced some temporary improvement. Neurologic examination gave negative results. Examination of the fundi revealed choking of the disks of 3 diopters on the right and of 4 to 5 diopters on the left. A week later a slightly positive Kernig sign developed. Although there were no localizing signs, the neurologist thought an abscess of the brain was present, probably in the right temporal lobe, and spinal puncture was advised against because of the danger that the abscess would rupture into the lateral ventricle. The choking of the disks remained the same, but hemorrhages and exudates appeared in the retinas. The condition remained unchanged, and three weeks later it was decided to explore the right temporal lobe and to make ventricular estimation. The patient was transferred to the care of the neurosurgeon.

Subtemporal decompression was done over the right temporal lobe. The dura was tense, and when it was incised there was a gush of clear fluid. A trochar was inserted through the superior temporal convolution, but an abscess was not encountered. The temporal horn was then entered, and 70 cc. of sanguineous fluid was removed. The same amount of air was injected for the purpose of

taking a ventriculogram, and the wound was closed. The roentgenogram of the head gave evidence of little air in the ventricle but of a great deal of air over the cortex, which, in the opinion of the neurosurgeon, made the presence of an abscess of the temporal lobe questionable.

The patient's headache, nausea and vomiting disappeared immediately after operation, and she became mentally clear. The temperature became normal on the second postoperative day and remained normal. The choking of the disks had receded to 2 diopters in each eye on the third postoperative day. She was dismissed on the tenth postoperative day with no choking of the disks and no other symptoms.

CASE 2.—A woman aged 22 reported that she had had high fever for a month. She had had scarlet fever a year previously. The left ear drum was slightly reddened, and when it was incised pus escaped. A roentgenogram was reported to reveal slight cloudiness of the left mastoid, with questionable destruction of the cells above the antrum. She was observed for two weeks; slight tenderness over the mastoid and slight concentric narrowing of the left external auditory canal developed. Except that it revealed slight secondary anemia, general physical examination gave negative results. She continued to have a high fever. All agglutination tests gave negative results, as also did inoculation of a guinea-pig with material obtained from the left ear. A diagnosis of mastoiditis was made, and mastoidectomy was advised.

At operation, an extensive small-celled mastoid process was noted. Little free pus was present, but all the cells were filled with hyperplastic granulation tissue. The patient's fever continued unabated, and a week later slight redness appeared in the right membrana tensa. This was incised, and a slight discharge followed. Examination of the eyes at that time gave completely negative results. Although a roentgenogram of the mastoids indicated no involvement on the right, in view of the course of the condition of the left ear exploration of the right mastoid was advised.

At operation the mastoid cells on the right seemed much broken down, with considerable pus and infected granulations throughout. The sigmoid sinus was torn, and the bleeding was controlled with a pack. The temperature dropped to normal after the operation but tended to rise suddenly at irregular intervals.

On the second postoperative day the patient complained of vertigo, but nystagmus could not be made out. On the seventh postoperative day she complained of severe occipital headache. Neurologic examination gave negative results. On the tenth postoperative day she complained of diplopia and was drowsy. Examination of the eyes disclosed weakness of the external rectus muscle on both sides, more marked on the left. There was choking of the disk of from 2 to 3 diopters on each side, with hemorrhages and exudates. On the twelfth postoperative day there was a sudden discharge of a large quantity of pus from the wound in the right mastoid, suggesting a discharge from an epidural abscess. Transient improvement followed this discharge. Neurologic examination at that time disclosed a plantar reflex of extensor type on the left side, a stiff neck, a positive Kernig sign and weakness of the sixth cranial nerve on each side. These evidences remained unchanged. Examination of the ocular fundi revealed no change in the choked disks. On the twenty-fifth postoperative day there was complete palsy of the sixth cranial nerve on both sides. No deep reflexes could be elicited, and there was slight facial weakness on the right. There was no change in the choked disks. On the thirty-fifth postoperative day there were a positive Babinski sign on the left side, some incoordination of the left hand, some weakness of the left arm and leg and slight weakness of the right side of the face. The neurologist was of the opinion that an abscess of the right temporal lobe was

present. The spinal fluid was clear and under moderately increased pressure, and the cytologic observations were normal. Exploration of the right temporal lobe was advised by the neurosurgeon.

At operation through an opening made for subtemporal decompression on the right side, the dura was found tense and bulging, and on incision of it a large quantity of clear fluid gushed out. An abscess was not found on exploration of the temporal lobe with a cannula.

The patient made a rather stormy postoperative convalescence, but the temperature became normal after two weeks, and she was dismissed from the hospital after the end of the third week.

Examination of the visual fields a month after the decompression disclosed a central relative scotoma on the left side. There were papilledema of 2 diopters in both eyes, pallor of both disks and a subhyaloid hemorrhage in the right eye. The patient had completely recovered three months later and was able to return to work.

Comment on Cases 1 and 2.—In the two cases just described there were no definite localizing signs, but in both the symptoms suggested an abscess of the temporal lobe. The spinal fluid was normal in both cases. There was considerable contrast in the rapidity of recovery in the two cases in favor of the one in which the ventricle was drained. Other factors may have played a part in this delayed recovery, but it is at least suggestive of a favorable influence from ventricular drainage. The rapidity with which the first patient recovered does not favor the hypothesis that nonsuppurative encephalitis was present.

CASE 3.—A boy aged 11 years was presented for examination with the history that earache on the left side had started after a cold three weeks previously. A week before his admission to the hospital the left membrana tensa had been incised, and this had been followed by a profuse purulent discharge. Since the myringotomy he had complained of frontal headache on both sides. In the evening of the day of admission he had a sharp chill, with a rise of temperature to 104 F., accompanied by drowsiness and rigidity of the neck.

On examination of the ears, nose and throat thick, pulsating pus was found in the left auditory canal. The membrana tensa was red and bulging. Tenderness could not be elicitied over the mastoid. On neurologic examination the patient was found to be stuporous; the neck was markedly rigid, and all tendon reflexes were almost absent. There was a positive Kernig sign. Spinal puncture revealed turbid fluid under moderately increased pressure. The spinal fluid contained 256 lymphocytes and 1,120 polymorphonuclear leukocytes per cubic millimeter. Organisms were not found on smear of the fluid, and a growth of organisms was not obtained on culture after forty-eight hours. Examination of the ocular fundi revealed relatively full retinal veins, but otherwise the results were negative. A diagnosis of leptomeningitis complicating acute mastoiditis was made, and exploration of the temporal bone was advised.

At operation the mastoid was found to be generally sclerotic, but here and there in the sclerotic bone were cellular portions containing infected granulation tissue and pus. There was a particularly marked involvement in the zygomatic root, and the dura overlying this area was reddened. The sigmoid sinus appeared normal. Two lines of diseased cells led around the labyrinthine capsules to the apex, one along the superior petrosal sinus and the other between the labyrinthine capsule and the jugular bulb.

The temperature fell to normal on the second day, with rapid subsidence of the signs of meningeal involvement. On the day following operation the cerebrospinal fluid contained 251 polymorphonuclear leukocytes, 368 small lymphocytes and 277 large lymphocytes per cubic millimeter. On the seventh postoperative day the temperature rose to 101 F. and rapidly fell to normal again without the patient's experiencing any sensation of chilliness. The neck became slightly rigid, and he was irritable and had photophobia. The fundi were normal. On the tenth day the wound in the mastoid was completely healed and the tympanic membrane was almost normal in appearance. There was slight nystagmus when he looked to the left and upward. Past-pointing with the left hand was present. He experienced considerable vertigo on moving the head; he had a severe headache, and vomiting was of the projectile type. The ocular fundi remained normal in appearance. The symptoms increased rather rapidly, and on the thirteenth postoperative day he lapsed into a semicoma. The eyes were deviated to the right, and he could not rotate them beyond the midline to the left. The left arm and leg were weaker than the right and were markedly ataxic. The pupils reacted sluggishly to light. He was very pale and had a rather weak, thready pulse of 58 beats per minute. His condition became rapidly worse during the day, and by 4 p. m. it seemed extremely critical. The neurologists and the neurosurgeon were of the opinion that there was an abscess in the left cerebellar lobe and that, judging from the history, the abscess had begun to form with the onset of the infection in the ear. Emergency exploration of the left cerebellar lobe was decided on, with the provision that if exploration of the cerebellum gave negative results the left temporal lobe should be explored. The left cerebellar lobe was explored from the posterior aspect; four punctures were made with the brain cannula, with negative results. Some cerebellar tissue was secured for microscopic study and was later reported to be normal. The left temporal lobe was then explored through an opening for temporal decompression, but an abscess was not found. The ventricle was then punctured, and a large quantity of clear cerebrospinal fluid, under tremendous pressure, shot out of the cannula. The condition was much improved immediately after the operation, and by the fourth day the patient was apparently normal except for slight residual ataxia in the left hand. He was dismissed on the fourteenth day following the ventricular drainage in apparently normal condition.

Comment on Case 3.—This was obviously a case of acute internal hydrocephalus. The absence of encephalitis was confirmed by microscopic examination of the cerebellar tissue removed. There was no evidence of a subdural collection of cerebrospinal fluid, but there was a large quantity under greatly increased pressure in the ventricle. It seems logical to assume, therefore, that there was a block in one of the foramina. That this was not attributable to fibrous adhesions seems evidenced by the lack of recurrence of symptoms. That ventricular puncture was a life-saving measure is the opinion of all concerned. The rapid recovery of this patient may also be contrasted with the slow postoperative course in those cases in which ventricular puncture was not done.

CASE 4.—A man aged 49 had had running ears since childhood. His mind was confused, and a history was difficult to obtain. For several years there had been a dull headache on the left, with occasional exacerbations of neuralgic pain running

from over the left eye to the left temporal region and along the left maxilla. This pain had been of increased severity for the preceding six months. The patient had lost 20 pounds (9 Kg.) in the preceding two months. On examination of the ears, nose and throat marked atresia of the left external auditory canal was found; the conchal opening was reduced to a mere slit filled with foul pus. The right tympanic membrane was scarred, thickened and retracted. Other findings referable to the ear, nose and throat had no bearing on the condition present.

On examination of the eyes small, contracted pupils were noted, which reacted sluggishly both to light and in accommodation; otherwise no abnormality was observed except for questionable pallor of the left optic disk. A roentgenogram of the mastoids gave evidence of partial sclerosis on both sides. A neurologic examination resulted in the diagnosis: "central nervous system objectively normal." However, the neurologist suspected the presence of localized serous meningitis, meningo-encephalitis or syphilis. The spinal fluid was clear, under normal pressure, contained 29 small lymphocytes to the cubic millimeter and reacted positively to tests for globulin. The Kline and Kolmer tests gave negative results. It was decided that elimination of the infection of the left ear should be the first step in treatment of this patient, and exploration of the left mastoid was advised. At operation a very sclerotic cortex was noted. The mastoid antrum was large and was filled with both cholesteatoma and infected granulation tissue. On removal of bone from the root of the zygoma, pus shot out under pressure and a huge epidural abscess was found extending from the region of the upper knee of the sigmoid sinus, anterior to the zygomatic root, well into the temporal fossa and up over the lateral surface of the temporal lobe to about 5 cm. above the level of the tegmen mastoideum. The bone over this abscess was removed to its limits; the dura was covered with thick granulations. It was thought that this finding adequately explained the patient's symptoms, and the dura of the left cerebellar lobe unfortunately was not exposed. He made an uneventful recovery except for occasional headache, but his memory was erratic. Examination of the central nervous system was repeated, and the findings were objectively negative. Examination of the eyes gave negative results; he was dismissed three weeks after the operation and was asked to return for observation in six months.

The patient returned in three months, saying that about two months after the operation his eyes had begun to feel as if they were jerking, the room seemed to twirl when he blew his nose and he carried a cane because of momentary sudden dizziness. A week before his return to the hospital severe pain developed in both eyes; there were some pain in and rigidity of the neck, and he vomited several times.

On examination of the ear, nose and throat, the cavity remaining after the radical operation on the left was seen to be completely epitheliated. On examination of the eyes, the fundi and visual fields were found to be normal. There was a moderate degree of nystagmus when the patient looked to the left, and there was slight limitation of the lateral gaze to the left. There were moderate ataxia of the left arm and leg, moderate nuchal rigidity and a bilaterally positive Kernig sign. He lay supine, with the eyes open, in a semicoma from which he could be aroused, but he appeared unable to comprehend questions. These observations suggested the syndrome of pontile meningitis suggested by Eagleton. A neurologic diagnosis of intermittent hydrocephalus with a cerebellar abscess on the left side was made. He was observed for three days, becoming increasingly more drowsy, and exploration of the left cerebellar lobe was advised by the neurosurgeon.

Cerebellar craniotomy was performed, and the left cerebellar lobe was explored with a cannula. Rubbery resistance was felt at a depth of 5 cm. The trochar

was pushed through this wall, and a small quantity of thick pus was evacuated; this was followed by a rather clear bloody fluid which greatly resembled sanguineous cerebrospinal fluid. The patient's reaction to the operation was not good, and he remained in a semistuporous condition. His condition became progressively worse, and he died on the fifth postoperative day.

At necropsy it was found that the lesion discovered had been a localized collection of purulent material in the subdural space. There was marked dilatation of the ventricles, which was thought to have been present for a long period, and on microscopic sections chronic arachnoiditis was observed.

Comment on Case 4.—This case illustrates the necessity of sufficiently extensive exploratory mastoidectomy when there is any question of the type of lesion present. It is conceivable that uncovering the cerebellar dura would have revealed changes suggestive of the lesion present. Drainage through the mastoid wound would have kept the meningitis localized and might have helped toward a successful outcome. The effect of the chronic encephalitis present in this case may have had a major bearing on the course of events. If ventricular drainage had been done when the neurologist suggested hydrocephalus, the part of the symptoms produced by the hydrocephalus would have been abolished and possibly a part, at least, of the stupor and the confused mental state would have been banished; in this way more time might have been afforded to arrive at a more nearly correct diagnosis. The localized, subdural abscess may be conceived as localized suppuration arising from an external hydrocephalic lesion. This case is certainly an instance of pseudo-abscess, although definitely to classify the condition as external hydrocephalus might be open to question.

IV. CONCLUSIONS

1. Otitic hydrocephalus is not a distinct entity but is a type of acute or subacute hydrocephalus produced by bacteria or toxins.

2. Its frequent association with otitic disease is attributable to the frequency with which the dura and the dural contents are involved by these lesions.

3. In certain instances the lack of pleocytosis in the spinal fluid may be the only finding which suggests the absence of an abscess of the brain. Pleocytosis may be present in acute hydrocephalus if there has been recent meningitis.

4. Ventricular puncture will relieve the condition and should be employed when response to spinal drainage is delayed or lacking.

5. Ventricular puncture is suggested for the acute hydrocephalus which accompanies encephalitis when the course is rapidly declining; this may relieve the symptoms of compression, may allow walling off of the encephalitis and may make possible selection of the optimal time for surgical intervention.

MENINGITIS DUE TO THE TYPE III PNEUMOCOCCUS

REVIEW OF THE LITERATURE AND REPORT OF A CASE OF OTITIC ORIGIN WITH RECOVERY FOLLOWING RADICAL MASTOIDECTOMY AND LABYRINTHECTOMY

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The high mortality from meningitis due to the type III pneumococcus shown by the scarcity of the recoveries reported in the literature makes one skeptical of the correctness of the diagnosis when a patient does recover. I think the diagnosis in the case reported here was well substantiated.

I found in the literature reports of four cases in which the condition was treated by various methods with recovery. The diagnosis seemed to be correct, as the type III pneumococcus was recovered from the spinal fluid.

Steinholz and Gleich [1] reported the recovery of a girl, aged 7 years, after the use of antipneumococcus serum intraspinally and intracisternally when the intraspinal route became blocked. They also used the serum intramuscularly. They expressed the belief that the child recovered because the infection was localized and the strain of the organism was attenuated.

Cavenagh [2] treated a patient on whom he performed a radical mastoidectomy and administered 15 cc. of antistreptococcus serum intrathecally before the culture showed type III pneumococci. After this he used antipneumococcus serum (type not given) intrathecally, and recovery occurred within five weeks. This patient had lobar pneumonia also.

Weinberg [3] reported the recovery of a patient treated with retention enemas of potassium permanganate, as advocated by Nott.[4] His patient was free from fever within eleven days.

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1. Steinholz, R., and Gleich, M.: Pneumococcus (Type III) Meningitis: Recovery, J. A. M. A. **105**:795 (Sept. 7) 1935.

2. Cavenagh, J. B.: Recovery from Pneumococcal Meningitis, J. Laryng. & Otol. **48**:337, 1933.

3. Weinberg, Max H.: Case of Pneumococcus (Type III) Meningitis Treated with Potassium Permanganate—Recovery: A Plea for Its Trial, J. Nerv. & Ment. Dis. **74**:38 (July) 1931.

4. Nott, H. W.: The Thyroid and Manganese Treatment in Various Diseases (First Report), Brit. M. J. **1**:443, 1925.

Ratnoff and Litvak [5] reported the cure of a girl, aged 7½ years. For the first five days they gave her antipneumococcus serum intraspinally without change in the clinical picture. At that time they started intraspinal injections of ethylhydrocupreine hydrochloride, in addition. There was immediate improvement, which continued for three days, at the end of which time the patient complained of blurred vision; the injections of ethylhydrocupreine hydrochloride were discontinued, but daily lumbar punctures were continued, with injections of 20 cc. of antipneumococcus serum. The symptoms cleared up completely, and the patient was discharged cured. They expressed the belief that ethylhydrocupreine hydrochloride enhances the curative value of antipneumococcus serum.

REPORT OF CASE

A. C., an Italian girl aged 15, was admitted to the Massachusetts Eye and Ear Infirmary on Feb. 27, 1931, with a history of intermittent discharge from the left ear for three years and constant discharge for the past year. Headache and occasional vomiting had been present for six days. The headache became more severe; some pain was noted in the left ear on the morning of admission, and by noon she was semiconscious and complained of pain in her back. No history of dizziness was elicited.

The past medical history and the family history were irrelevant. Local examination showed the right ear to be normal. The left external auditory canal was filled with polyps and foul pus. No response to the caloric test could be elicited in the left ear.

Neurologic examination revealed absence of nystagmus, normal eyegrounds, increased reflexes, a positive Kernig sign and a stiff neck.

Laboratory examination showed 24,000 white blood cells. The urine was normal.

Lumbar puncture revealed cloudy fluid under a pressure of 180 mm. of mercury, with 9,000 cells, 88 per cent of which were polymorphonuclear leukocytes. No organisms were noted in the smear. Culture of the cerebrospinal fluid showed type III pneumococci on March 1.

A roentgenogram showed a cholesteatoma in the left mastoid antrum; the remainder of the mastoid was sclerosed.

On February 27 radical mastoidectomy and labyrinthectomy on the left side were performed with the patient under ether anesthesia. The mastoid cells were completely sclerosed except in the region of the antrum, where a large cholesteatoma was found, which extended into the cavity of the middle ear. This had eroded through the wall of the horizontal semicircular canal, forming a fistula. The facial nerve was found to be exposed by disease and enmeshed in granulations throughout its course across the inner wall of the cavity of the middle ear.

After the cholesteatoma and the cavity of the middle ear were cleaned out, the labyrinth was opened and thin pus escaped.

On February 28 the patient's condition remained the same and she was still unconscious. Lumbar puncture yielded fluid with the appearance of ground glass and under a pressure of 350 mm. of mercury; the cell count was 2,160.

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5. Ratnoff, H. L., and Litvak, A. M.: Pneumococcus Meningitis Treated with Morganrath's Optochin Hydrochloride, Arch. Pediat. **43**:466 (July) 1926.

On March 1 the patient was conscious, with a slight headache, some dizziness and vomiting. Lumbar puncture yielded fluid under a pressure of 220 mm. of mercury, with 250 cells.

On March 3 the patient was improved and had less headache. The white blood cells numbered 11,500. Culture of blood taken two days before showed no organism. Culture of material from the mastoid showed gram-positive diplococci. The spinal fluid showed 110 cells.

Lumbar puncture was performed daily until March 5.

Culture of spinal fluid taken on March 1 showed type III pneumococci.

On March 5 a culture of spinal fluid taken two days before showed no growth. Less headache and no vomiting were noted. The condition was so much improved that it was decided to do no more lumbar punctures.

The patient continued to improve from day to day and was completely free from symptoms except for occasional headache and some nystagmus to the right side. This nystagmus continued for two months.

The patient was discharged to go home on April 8, forty days after admission; she was free from symptoms, except for a discharge from the cavity made at operation. This discharge has persisted for six years at the time of writing, but no other symptoms have developed, and she is working daily.

COMMENT

Of these five patients, four received subarachnoid drainage by lumbar or cisternal puncture or both. Two received antipneumococcus serum alone, one received antipneumococcus serum and ethylhydrocupreine hydrochloride and one received no subarachnoid therapy.

There seems to be no definite plan of treatment of this highly fatal disease except by an attempt to eliminate by surgical means any focus which may be found about the sinuses or the temporal bone. In most of the cases meningitis due to the type III pneumococcus seems to originate from the middle ear or the mastoid.

Instances of recovery from different types of pneumococcic meningitis after one or more lumbar punctures without other treatment have been reported. These (and probably the cases recorded here) were undoubtedly instances of invasion of the meninges by organisms of low virulence in a person with good resistance or immunity to that particular strain of organism, and the lumbar drainage has aided in dispersion of the infection. But in other cases, in which there is abundant plastic exudate which encapsulates foci in the subarachnoid space and thus constantly acts as a source of reinfection, spinal drainage alone could not be of much use. On the other hand, it might cause a spreading of the infection throughout the subarachnoid area.

In view of the fact that the subarachnoid space may harbor foci for reinfection of the meninges, it seems that the main objective, as shown by Stewart [6] in his experiments on meningitis in dogs, is to clear the

6. Stewart, F. W.: Local Specific Treatment of Experimental Pneumococcus Meningitis, J. A. M. A. **89**:1316 (Oct. 15) 1927.

subarachnoid area of the plastic exudate and to bring the therapeutic agent into direct contact with all areas of the meninges. Stewart accomplished this by quadruple puncture (lumbar and cisternal puncture and bilateral trephining) and injection of ethylhydrocupreine hydrochloride and serum mixtures. Seemingly a heroic procedure, but undoubtedly justifiable in consideration of his results with desperately ill patients!

From a review of the literature, not only on meningitis due to the type III pneumococcus but on the other types of pneumococcic meningitis, and the comments of the different writers on the subject, I think the following points are important:

1. Early diagnosis and identification of the organism.
2. Elimination of the focus by surgical means.
3. Early administration of highly concentrated serum.
4. Measures to make sure that whatever the therapeutic agent, it shall be made to reach all areas of the subarachnoid space.

MENINGITIS DUE TO THE TYPE IV PNEUMOCOCCUS, WITH RECOVERY

REPORT OF A CASE

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In 1935 Tripoli [1] made a study of the therapeutic procedures for bacterial meningitis at the New Orleans Charity Hospital. In the previous ten years there were 111 cases of pneumococcic meningitis, in which treatment had been given by almost every known method. Of the 111 patients 110 died. The sole survivor had been treated with antimeningococcus serum by substitution.

In a survey of the literature for the past fifteen years I was able to find some 30 other cases in which recovery followed various procedures. Though each author stanchly advocates his own method, one has the feeling that the 30 cases when compared with the thousands of deaths from pneumococcic meningitis would give an average similar to the statistics of Tripoli, the only procedure showing any consistent results being the use of specific serum in cases in which the condition is amenable to it.

It has been our experience with these methods alone, in combination with each other or with bizarre additions of our own, that the results have been uniformly unsuccessful.

In reporting a single case of pneumococcic meningitis with recovery we wish to add a dramatic case to the rather sparse literature, and in the words of many of the previous reports of single cases, we should like to advocate the procedure stanchly, with the hope that further experience will prove it to be of value.

REPORT OF CASE

F. D., a girl aged 13, had what was considered a mild attack of "influenza" one week before admission to the hospital. It confined her to the house with a temperature from 99 to 100 F. Six days before admission she complained of pain in the left ear. Four days before admission she had a mild headache in

1. Tripoli, C. J.: Bacterial Meningitis: Comparative Study of Various Therapeutic Measures, J. A. M. A. **106**:171-177 (Jan. 18) 1936.

the left temporal region and severe pain in the left ear. Paracentesis revealed pus under pressure. On the following day she had a marked rise in temperature, with violent headache and a chill lasting thirty minutes but no vertigo. The condition rapidly became worse, and she was admitted to St. Margaret's Hospital on Feb. 23, 1937.

Past History.—The patient had always been well, having won several prizes at school for "perfect health."

Physical Examination.—When first seen by us the child seemed almost moribund; the temperature was 105 F., the pulse rate 110 and the respiratory rate 40. She was irrational and in opisthotonos. The neck was rigid, and the Kernig sign was positive. She was beginning to show mottled cyanosis of the extremities, and the respiration was weak and irregular.

Examination showed a scant gray discharge from the left ear drum, which was thick and boggy. There were no postaural signs, and examination of the ear, nose and throat otherwise gave negative results. The fundi and the ocular movements were normal, and the remainder of the gross neurologic examination gave negative results.

The heart and lungs were substantially normal, as confirmed by roentgenograms. A roentgenogram of the left mastoid showed diffuse thickening, with beginning destruction of the cells of the tip.

Laboratory examination by Dr. Trumper revealed a white blood cell count of 14,000, with 84 per cent polymorphonuclears, and a hemoglobin content of 78 per cent.

Lumbar puncture showed cloudy fluid under increased pressure; it contained 2,000 cells, all polymorphonuclears, and an occasional gram-positive diplococcus was seen on smears. Culture on dextrose serum and brain-heart infusion showed a pure growth of pneumococci, which were gram-positive, encapsulated and soluble in bile. A Neufield reaction carried out for types I, II, III, V, VII, and VIII showed that the organism was not of these types. Agglutination tests with rabbit serum for types I, II and III confirmed this. The culture was avirulent for white mice three days later. Culture of the blood gave negative results.

On the theory originally held that the organism would prove to be a streptococcus, the administration of prontosil,[2] 10 cc. every six hours, was started.

A simple mastoidectomy was done on the left side, with extensive exposure of the dura over the temporal lobe and the cerebellum. There was early destruction of a few cells, and pus was noted in many. The dura was tense but did not seem inflamed. On the following day the condition was improved slightly, and the administration of prontosil was continued; but since the organism proved to be an untyped pneumococcus, sulfanilamide, 5 grains (0.3 Gm.) every four hours, was added to the prontosil on the third postoperative day.

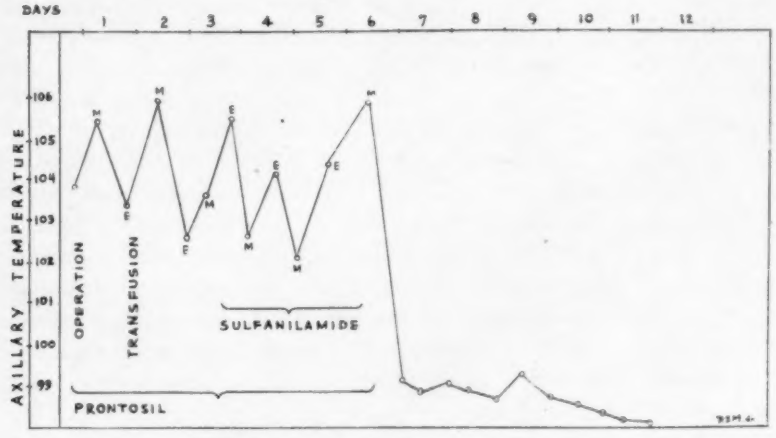
A small blood transfusion was given, and some one suggested giving a small amount of solution of formaldehyde U. S. P. intravenously. So far as could be observed, neither of these had any appreciable effect, and they were not repeated.

On the sixth postoperative day the condition became much worse. The child went into deep coma; the respiration was very irregular and the pulse

2. The term prontosil has been used for a number of substances. As used in this paper it refers to the disodium salt of 4′-sulfamidophenyl-2-azo-acetylamino-1-hydroxynaphthalene-3, 6-disulfonic acid.

barely palpable, and the temperature rose to 105.8 F. All medication was discontinued, and an immediately fatal prognosis was given. Three hours later the child suddenly woke up, asked for a pickle and said that her head felt fine. The temperature dropped by crisis to normal and remained there. From that time on the most careful mental, physical and neurologic examinations have failed to reveal the slightest evidence of the recent condition. The mastoid healed rapidly, and she is being carefully followed for any complications.

Comment.—While removal of the apparent focus of infection after organisms have appeared in the spinal fluid is locking the door when the horse is already gone, we agree with Shambaugh,[3] Weil [4] and others that it is an essential part of the procedure in preventing further infection.



Temperature chart showing the postoperative fluctuations and the drop by crisis to normal on the sixth day.

It was shown by Marshall, Emerson and Cutting [5] that para-aminobenzesulfonamide (sulfanilamide) after oral administration appears in the spinal fluid in therapeutic amounts, and experimental work of Rosenthal [6] on mice has shown that it has a definite protective value

3. Shambaugh, G. E., Jr.: The Surgical Treatment of Meningitis of Otitis and Nasal Origin, J. A. M. A. **108**:696-700 (Feb. 27) 1937.

4. Weil, C. K.: Meningitis Due to Type I Pneumococcus: Report of Case with Recovery Due to Serum, Arch. Int. Med. **57**:514-520 (March) 1936.

5. Marshall, E. K., Jr.; Emerson, K., Jr., and Cutting, W. C.: Para-Aminobenzene Sulfonamide: Absorption and Excretion, J. A. M. A. **108**:953-957 (March 20) 1937.

6. Rosenthal, S. M.: Chemotherapy of Experimental Pneumococcus Infections, Pub. Health Rep. **52**:48-53 (Jan. 8) 1937.

against many virulent strains of the pneumococcus. On the other hand, he showed at the same time that prontosil has little if any value against this organism.

The final drop in temperature strongly resembled a pneumonia crisis.

In closing, we can say only that a previously healthy child recovered from meningitis due to a type IV pneumococcus of low virulence after the administration of prontosil and sulfanilamide. Experimental work seems to confirm the value of the latter drug against pneumococci and justifies its further use in so desperate a condition.

THROMBOPHLEBITIS OF THE LATERAL SINUS

OBSERVATIONS IN A SERIES OF TWENTY-TWO CASES

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The greatest achievement one hopes for in any surgical procedure is to lessen mortality and morbidity. In proportion to this only does the surgical technic advocated receive the approbation of the general medical public. One can truly attest to such an accomplishment in the diagnosis and treatment of thrombophlebitis of the lateral sinus secondary to otitic disease.

It is my purpose in this paper to confine myself exclusively to those observations which in the main have not been stressed sufficiently in the more recent literature. I shall refrain from reiterating those essential points which are so often repeated and which long ago have been universally accepted as fundamentally essential in both the diagnosis and the treatment. It is only by calling attention to the rare and unusual that new data are contributed to otologic experience.

Sinus thrombosis may and does only too often assume a protean nature, and it is by possessing a perspective that is wide enough to take into view these atypical forms that the diagnostic acumen of the otologist is sharpened sufficiently to enable him to recognize them when they occur.

INCIDENCE AND MORTALITY

The incidence of sinus thrombophlebitis as a complication of otitic disease has been recorded by many observers. From a careful analysis of a great number of reports published both in this country and abroad, two important observations have been made. First, the more recent statistics show a definite diminution in the number of cases of otitic disease presenting this complication as compared with those of two or three decades ago. This is easily explained. With a better concept of the pathologic picture, diagnosis and treatment of mastoid disease in general, it follows as a natural consequence that complications emanating therefrom would diminish in number. Second, in series of cases which were studied in clinics and institutions, sinus thrombophlebitis was present two or three times more frequently than it was in

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series in which the patients were observed in private practice. This second observation is easily accounted for. Patients attending clinics are inclined to go for a much longer period without seeking medical aid. They therefore come to operation later and are more prone to those complications which in most instances result from extension of the infectious process by continuity and contiguity. Private patients seek medical aid sooner, are under more careful and constant observation and come to operation earlier.

Lillie,[1] in a series of 500 mastoidectomies at the Mayo Clinic, found sinus thrombosis as a complication in 3.8 per cent. Meltzer,[2] in a series of more than 4,000 mastoidectomies performed at the Massachusetts Eye and Ear Infirmary, recorded involvement of the lateral sinus in 3.5 per cent. Mygind[3] recorded the occurrence of 199 cases of sinus thrombosis in a series of 3,000 mastoidectomies, an incidence of almost 7 per cent. This is an unusually high figure, but it is more or less in keeping with the statistics gathered from foreign clinics, in which intracranial complications in general are observed far more often than in American institutions.

The mortality rate in sinus thrombosis was given generally as 50 per cent. Heine[4] at one time reported a mortality rate as high as 57 per cent. This, however, has been materially lowered in more recent years. Atkins[5] recorded fatalities in 37.5 per cent of cases; Coates, Ersner and Persky[6] in 35.7 per cent; Meltzer[2] in 27.8 per cent; Boies[7] in 25 per cent, and Brunner[8] in 22 per cent.

The observations herein set forth are based on an experience with 22 cases of thrombophlebitis of the lateral sinus. They have been taken from a series of 1,257 mastoidectomies performed between 1920 and 1936. The incidence of sinus involvement in this series reached only

1. Lillie, H. I.: Infection of Sigmoid and Lateral Sinus, Surg., Gynec. & Obst. **35**:418, 1922.

2. Meltzer, P. E.: Treatment of Thrombosis of Lateral Sinus: Summary of Results Obtained During Twelve Years at Massachusetts Eye and Ear Infirmary, Arch. Otolaryng. **22**:131 (Aug.) 1935.

3. Mygind, S. H.: Treatment of Otogenic Sepsis and Sinus Thrombosis, Acta oto-laryng. **16**:474, 1931.

4. Heine, B.: Operationen am Ohr. Die Operationen bei Mittelohreiterungen und ihren intrakraniellen Komplikationen, Berlin, S. Karger, 1913.

5. Atkins, R. T.: Report of Forty Cases of Lateral Sinus Thrombosis, New York State J. Med. **24**:885, 1924.

6. Coates, G. M.; Ersner, M. S., and Persky, A. H.: Lateral Sinus Thrombosis with Review of Literature, Ann. Otol., Rhin. & Laryng. **43**:419, 1934.

7. Boies, L. R.: Lateral Sinus Thrombosis: A Review of One Hundred and Eighty-Four Cases with Special Reference to the Postoperative Type, Ann. Otol., Rhin. & Laryng. **41**:227, 1934.

8. Brunner, Hans: Postoperative Sinus Thrombosis, Arch. Otolaryng. **10**: 217 (Sept.) 1929.

1.7 per cent of cases, a much lower rate than is generally recorded. It should be remembered, however, that this series constitutes only patients seen in private practice, a fact which is important in a study of the incidence of involvement of the sinus in otitic disease.

Of the 22 patients with thrombophlebitis of the lateral sinus, 19 recovered and 3 succumbed, the mortality rate being 13.5 per cent. Despite the fact that the number of cases is not as large as in some of the other series that have been reported, nevertheless, the series is composed of a sufficiently varied group of cases to enable one to evaluate the type of therapy employed, with its resultant efficacy.

RELATIONSHIP BETWEEN MASTOID DISEASE AND SINUS THROMBOSIS

Another interesting observation in connection with sinus thrombosis is the relationship of its occurrence to the prevailing type of mastoid infection. During certain years, when the number of mastoid operations performed was greater, there were fewer patients presenting this complication. On the other hand, during some years, with fewer mastoid operations, there were relatively more cases of sinus involvement. In other words, there was no constant ratio between the number of mastoid operations and the number of sinus complications resulting therefrom. A definite relationship, however, does seem to exist between the two so far as the type and severity of the mastoid infections are concerned. It is a notable fact that in those patients whose otitic disease is severe and rapid and leads to extensive and widespread softening and destruction of the mastoid cells, sinus complications are more likely to be encountered. Sinus disease depends primarily on the accessibility of the infection to the sinus walls. In these cases the sinus plate often shares in the bony disintegration, so that the sinus wall itself lies exposed to the invading organisms without the usual protection afforded it by the intact bony plate. On the other hand, when the prevailing otitic infection is not so rapid and fulminating, complications are not encountered so frequently. It is this fact, I believe, which accounts often for the apparent wide differences noted in the statistics recorded by some observers.

RELATIONSHIP OF THE STRUCTURE OF THE MASTOID TO SINUS DISEASE

It is further observed that there seems to exist some relationship between the structure of the mastoid and the occurrence of sinus disease. It can hardly be considered coincidental that a careful review of the operative charts of the 22 patients revealed that 17, or 77 per cent, presented mastoids that were small and in the majority of cases

Case; Age; Sex; Side	Brief Otitic History	Type of Mastoid; Operative Findings	Condition of Sinus	Advent of Symptoms Suggestive of Sinus Involvement	Temperature, F.	Diagnosis of Sinus Involvement	White Cell Count, Thousands	Blood Culture After Operation
1 11 yr. M Right	Pain in ear 10 days; no discharge; tenderness; sagging of canal; surgical mastoid	Small diploic forward sinus; free pus; sinus plate necrotic	Sinus wall diseased, thickened and discolored	Sinus involvement not suspected	Not septic; 103	At primary operation	16	1st —
2 9 yr. M Right	Otorrhea 13 days; pain; fever; tenderness; sagging of canal wall; surgical mastoid	Pneumatic; cells soft; free pus and granulation; sinus wall normal, emissary diseased and thick	Normal sinus; diseased emissary vein	Third postoperative day	Septic 99-105	Postoperative	11 14 17	2d —
3 7 yr. F Right	Incision of drum 4 weeks; fever; no pain or tenderness; resolving middle ear; mastoid not surgical	Small diploic mastoid; few cells; no free pus; sinus plate unbroken	Perisinuous abscess; sinus thickened and detached from sinus groove	4 days prior to mastoid operation	Septic; 100-106	Preoperative	19 23 22	3d —
4 4 yr. M Left	Drum incised; otorrhea 1 week; pain; tenderness; fever; sagging of canal; surgical mastoid	Small, sclerotic; few cells; free pus; granulation; sinus plate intact	Wall congested	3 days after mastoid operation	Continued high; 103-105	Postoperative	22 28	None —
5 3 yr. F Left	Incision of drum; discharge 10 days; later dry middle ear; headache; septic temperature; mastoid not surgical	Small shallow mastoid; diploic; few cells; no free pus; plate intact	Wall thickened; covered with granulations	4 days prior to mastoid operation	Septic; 101-104	Preoperative	12	3d —
6 6 yr. M Right	Otorrhea 4 weeks; tenderness; sagging; low grade fever; surgical mastoid	Large cellular mastoid coalesced; free pus and granulations; plate necrotic; perisinuous abscess	Wall thickened; covered with diseased granulations	21 days after mastoid operation	Septic; 99-105	Postoperative	21 18	1st — 2d — 3d + 4th —
7 9 yr. M Right	Otorrhea 8 days, pain; fever; tenderness; sagging; surgical mastoid	Small diploic; cells soft; free pus; plate necrotic	Wall diseased; covered with thick discolored granulations	Third day after mastoid operation	Septic; 100-105	Postoperative	18 24	10th —
8 8 yr. M Left	Earache 5 weeks; discharge 1 week; drum flat; middle ear resolving; mastoid not surgical	Small flat sclerotic mastoid; no free pus; sinus plate broken; perisinuous abscess	Wall thickened and covered with diseased discolored granulations	5 days before operation	Low grade sepsis; 100-102	Preoperative	18	2d — 3d — 4th + 5th + 6th —
9 4 yr. F Right	Otorrhea 2 weeks; pain; tenderness; septic temperature; no sagging; surgical mastoid	Small cellular mastoid; free pus and granulation; sinus plate necrotic	Wall diseased; many discolored granulations	4 days prior to operation	Septic; 100-106	Preoperative	19 21 26	1st —
10 18 yr. M Right	Otorrhea 2 weeks; pain; tenderness; sagging of canal wall; surgical mastoids	Large pneumatic mastoid; free pus and granulations; sinus plate unbroken	Wall diseased and thickened	4 days after operation	Septic; 100-106	Postoperative	18.5 29	2d —
11 14 yr. M Right	Otorrhea 3 weeks; pain; tenderness; sagging of canal wall; surgical mastoid	Small cellular mastoid; free pus in cells; sinus plate necrotic	Wall thickened; covered with diseased granulations	5 days after mastoidectomy	Septic; 100-106	Postoperative	21 28 29.5	5th —
12 7 yr. F Right	Otorrhea 10 days; no pain; no tenderness; slight discharge; no sagging; mastoid not surgical	Small diploic mastoid; few cells; softening slight; no free pus; sinus plate intact	Wall thickened; covered with many diseased granulations	4 days after mastoidectomy	Septic; 98-104	Postoperative	19 21	1st —
13 18 yr. F Right	Otorrhea 4 weeks; copious discharge; marked changes in canal; no pain; no tenderness; mastoid surgical	Large cellular mastoid completely broken down; free pus and granulations; sinus plate necrotic	Wall diseased, with thick soft granulations	3 days after mastoidectomy	Low grade; 99-101	Postoperative	88 85	1st —
14 5½ yr. F Right	Otorrhea 3 weeks; pain; tenderness; sagging of canal wall; surgical mastoid	Small; diploic; few cells; free pus and granulations; sinus plate intact	Diseased; covered with granulations; perisinuous abscess	4 days prior to mastoidectomy	Septic; 100-106	Preoperative	11 11	None
15 14 yr. M Left	Otorrhea 2 weeks; headache; no tenderness; no sagging of canal wall; mastoid not surgical	Mastoid small; few cells; little softening; no free pus; sinus plate necrotic	Wall inflamed; fine granulations on surface	2 days prior to operation	Septic; 100-104	Preoperative	10 12	1st —

* All were cured except patients in cases 4, 14 and 19, who died.

Blood Culture After Operation	Organism	Fundus	Type of Surgical Procedure	When Employed	Findings in Interior of Sinus	Transfusions	Other Symptoms
1st —	None	Not examined	Ligation of jugular vein; drainage of lateral sinus	Same time as mastoidectomy	Free pus and organized thrombi	None	Headache
2d —	Str. haemolyticus	Normal	Ligation of jugular vein above facial vein; drainage of lateral sinus	9 days after mastoidectomy	Obliterated emissary vein; thrombi; free bleeding from both ends	4	Chills, headache, vertigo
3d —	Str. haemolyticus	Normal	Ligation of jugular vein; opening of lateral sinus	Same time as mastoid operation	Thrombi; free bleeding from torcular end only	3	Chills and persistent vomiting, 4 days
None —	Nonhemolytic streptococcus	Normal	Ligation of jugular and facial veins; opening of sinus; washing out of bulb	Sinus open 9 days after mastoid; ligation 2 days later	No thrombi; free bleeding from both ends	6	Chills; cough; bloody expectoration
2d —	Str. haemolyticus	Normal	Ligation of jugular and facial veins; sinus drained	Same time as mastoid operation	Organized thrombi; bleeding from torcular end only	5	Chills; vomiting; pyelitis
1st — 2d — 3d + 4th —	Str. haemolyticus	Normal	(1) Ligation of jugular vein; sinus drained; (2) exposure of sinus to torcular, sinus wall excised	22 days after mastoidectomy; 9 days after ligation	Thrombi; free bleeding from both ends	4	Chills; vomiting; recurrence of sepsis
10th —	Str. haemolyticus	Fulness of retinal veins	Ligation of jugular vein; drainage of sinus	6 days after mastoid operation	No thrombi; free bleeding from both ends	10	Chills
2d — 3d — 4th + 5th + 6th —	Str. haemolyticus; nonhemolytic streptococcus	Blurring of disk margins, bilateral	(1) Ligation of jugular and facial veins; opening of sinus; (2) exposure of sinus to torcular; excision of sinus wall	Ligation at same time as second mastoid operation 11 days later	Free pus and organized thrombi; free bleeding from bulbar end only	6	Chills; Kernig sign, both sides; severe headache; recurrent sepsis
1st —	None	Normal	Ligation of jugular vein above facial vein; opening of lateral sinus	Same time as mastoid operation	Organized thrombi; free bleeding from both ends	3	Chills
2d —	Nonhemolytic streptococcus	Fulness of retinal veins, bilateral	Ligation of jugular vein below facial vein; opening of lateral sinus	4 days after mastoidectomy	Thrombi; free bleeding from both ends	4	Chills; vomiting
5th —	Str. haemolyticus	Choked disk, 2 diopters, noted after ligation of jugular vein	Ligation of jugular and facial veins; opening of sinus	9 days after mastoidectomy	Thrombi; bleeding from torcular end only	4	Chills; painful deglutition; headache
1st —	Str. haemolyticus	Blurring of disk margins, more on right	Ligation of jugular vein above facial vein; opening of sinus	6 days after mastoidectomy	Thrombi; free bleeding from both ends	3	Chills; headache
1st —	Str. haemolyticus	Swelling of both disks to 2 diopters	Ligation and excision of jugular vein from above facial vein to level of clavicle; opening of lateral sinus	4 days after mastoidectomy	Organized thrombus reaching from torcular through bulb into vein to clavicle	None	Chills; diplopia; severe vertigo
None	Str. haemolyticus	Normal	Ligation of jugular vein above facial vein; opening of sinus	Same time as mastoid operation	Thrombi; free bleeding from bulbar end only	None	Patient did not come out of anesthesia
1st —	Str. haemolyticus	Normal	Ligation of jugular and facial veins; opening of sinus	Same time as mastoid operation	Large organized thrombus; free bleeding from both ends	2	Persistent vomiting; rigidity of neck; spinal fluid showed 250 lymphocytes

Case; Age; Sex; Side	Brief Otitic History	Type of Mastoid; Operative Findings	Condition of Sinus	Advent of Symptoms Suggestive of Sinus Involvement	Temperature, F.	Diagnosis of Sinus Involvement	White Cell Count, Thousands	Blood Culture After Operation
16 7½ yr. M Left	Otorrhea 7 days; headache; no tenderness; no changes in canal; mastoid not surgical	Mastoid small, with few cells; pus and granulations in antrum; sinus plate intact	Wall thickened, discolored and covered with abundance of granulations	3 days after mastoidectomy	Septic; 100-105	Postoperative	22 24	1st — 2d — 3d + 4th + 5th —
17 9½ yr. M Right	Otorrhea 6 days; pain 2 weeks; slight tenderness; no changes in canal; mastoid not surgical	Mastoid small and diploic; free pus in antrum; sinus plate necrotic; perisinuous abscess	Wall thickened, with discolored granulations; perforation in wall	3 days prior to operation	Septic; 101-105	Preoperative	23	1st —
18 5 yr. M Right	Earache 2 weeks; discharge 10 days; drum flat; no sagging; no tenderness fever; mastoid not surgical	Mastoid small, with few cells; free pus and granulations in tip; sinus plate necrotic; perisinuous abscess	Diseased and thickened; small area or necrosis at knee of sinus	3 days prior to mastoidectomy	Septic; 98-106	Preoperative	11	1st —
19 12 yr. F Left	Pain in ear 3 weeks; otorrhea 2 weeks; tenderness; sagging of canal; mastoid surgical	Mastoid large and cellular; marked destruction; free pus and granulations; sinus plate necrotic; perisinuous abscess	Wall thickened and covered with diseased granulations	3 days after mastoidectomy	Low grade sepsis; 100-102	Postoperative	14 12 9.6	None —
20 8 yr. M Right	Discharging ear 3 weeks; no tenderness; no sagging of canal wall; mastoid not surgical	Mastoid small and diploic; some softening at tip; no free pus; sinus plate intact	Wall normal	3 days prior to mastoidectomy	Septic; 100-104	Postoperative	14 16	2d —
21 7½ yr. F Left	Discharging ear 3 weeks; tenderness; pain; sagging of canal wall; mastoid surgical	Mastoid small and diploic; markedly softened; free pus and granulations; sinus plate destroyed; perisinuous abscess	Wall thickened and covered with discolored granulations	3 days after mastoid operation	Septic; 99-105	Postoperative	16 22 24	2d —
22 14 yr. M Left	Discharging ear since childhood; acute exacerbation 5 days before admission to hospital; discharge foul; central perforation; mastoid surgical	Mastoid small and sclerotic; free pus and granulations; sinus plate necrotic; sinus wall covered with granulations	Wall thickened and covered with granulations	3 days prior to mastoid operation	Septic; 99-104	Preoperative	15 16	3d —

diploic. In only 5 instances was the mastoid pneumatic. It must seem obvious, therefore, that in the former group there exists something in the anatomic structure and arrangement which renders this particular type of mastoid more prone to the development of sinus disease. There exists the undisputed clinical fact that the right lateral sinus becomes implicated far more often than the left. In this series of 22 cases the right sinus was affected in 13. This is based on the well established anatomic fact that the right sinus approximates more closely to the posterior canal wall, thus bringing it within easier reach of the invading organisms. In the large broad pneumatic mastoid the sinus must of necessity be situated at a greater distance from the initial site of the mastoid infection, e. g., the antrum. On the other hand, in the small mastoid, the posterior boundary of which is the sinus plate, the sinus is brought into closer proximity to the posterior canal wall, and in this location it becomes more vulnerable. It is interesting to note that in almost every case in which the mastoid was small, operation disclosed a perisinuous abscess at the primary mastoid operation. The conclusion to be drawn, therefore, is that the small mastoid is more prone to the development of involvement of the sinus. Furthermore, in the majority of instances the sinus becomes involved as a result of direct extension

Cases of *of the Lateral Sinus*—Continued*

White Cell Count, Thousands	Blood Culture After Operation	Organism	Fundus	Type of Surgical Procedure	When Employed	Findings in Interior of Sinus	Transfusions	Other Symptoms
22 24	1st — 2d — 3d + 4th + 5th —	Str. haemolyticus	Normal	Ligation of jugular vein above facial vein; opening of sinus	6 days after mastoid operation	No thrombus; free bleeding from both ends	6	Chills; headache
23	1st —	Str. haemolyticus	Swelling of right disk to 2 diopters; blurring of margins on left	Ligation of jugular and facial veins; excision of jugular vein; sinus opened	Same time as mastoid operation; ligation 24 hours later	Well organized thrombus; pus oozing from sinus; free bleeding from torcular end only; disease of jugular vein	5	Headache; rigidity of neck; vomiting
11	1st —	Str. haemolyticus	Normal	Ligation of jugular and facial veins; sinus opened	Same time as mastoid operation	Free pus; many well organized thrombi; free bleeding from torcular end only	4	Chills; headache
14 12 9.6	None —	Str. haemolyticus	Normal	Ligation of jugular vein below facial vein; sinus opened	Lateral rupture of sinus 9 days after mastoid operation; ligation 7 days after	No thrombus; free bleeding from both ends	5	Chills; headache; cardiac dilatation
14 16	2d —	Str. haemolyticus	Blurring of both disks	Ligation of jugular vein below facial vein; obliteration of lateral sinus	5 days after mastoid operation	Large thrombus in bulbar end; free bleeding from torcular end	2	Severe headache
16 22 24	2d —	Str. haemolyticus	Normal	Ligation of jugular vein below facial vein and obliteration of lateral sinus	5 days after mastoid operation	Large well organized thrombus in torcular end; free bleeding from both ends	2	Chills; headache
15 16	2d —	Str. haemolyticus	Normal	Ligation of jugular vein and obliteration of lateral sinus	Same time as mastoid operation	Free pus in lateral sinus; many organized thrombi removed; slight bleeding only from torcular end	4	Chills; headache; sweats

of the infection from the mastoid cells to the sinus plate by continuity. The deciding factors are: first, the rapidity with which the infection spreads, this being governed by the virulence of the organisms, and second, the distance it must travel to reach the sinus, this being governed to a great extent by the size of the mastoid process.

SYMPTOMATOLOGY

There is little that can be added to what is already known and has many times been repeated concerning the symptomatology of thrombophlebitis of the lateral sinus. In general, it may be said that the diagnosis in the majority of instances offers no serious or complicated problem. However, it may be noteworthy that extensive disease of the lateral sinus may exist without any local or general evidence of its presence.

In case 1,[9] there was a history of otitis of only ten days' duration. The drum was intact, and there was no aural discharge. The patient walked into my office complaining of headache. He gave no history of chills or fluctuating temperature.

9. Greenfield, Samuel D.: An Unusual Case of Lateral Sinus Thrombosis, M. J. & Rec., December 1924.

Operation disclosed an extensively diseased and necrotic sinus in which free pus was demonstrated. Patient 13 had a well organized thrombus that extended from the torcular Herophili through the lateral sinus and the jugular bulb into the greater portion of the jugular vein, reaching almost to the clavicle. Yet the patient slept well, never had a temperature above 101 F. and visited my office complaining of moderate headache and an annoying otorrhea of five weeks' duration.

Temperature.—Of all the symptoms present in thrombophlebitis of the lateral sinus, a septic temperature seems to be both the commonest and the most significant. At least, it is usually the first symptom that causes concern. In all but 3 cases of this series, the occurrence of a septic temperature was the first indication that the sinus was involved. The fluctuations were wide in most instances, the difference between the maximum and the minimum temperature being as much as six or seven degrees. The suddenness of the rise and fall were especially noted, and great significance should be attached to this phase. In 2 cases the temperature curve was atypical. In case 4 the temperature remained persistently high, ranging between 103 and 105 F. In case 13 it remained persistently low, ranging between 99 and 101 F.

Chills.—In 15 cases of this series chills preceded the rise in temperature. This was particularly noted in some of the younger patients despite the fact that chills are not expected in the very young. In the absence of other sources that would satisfactorily explain the cause of the pyrexia in all patients presenting chills and fever in the presence of otitic infection, a diagnosis of sinus disease was ventured.[10] It is interesting to note, therefore, that on these symptoms alone a preoperative diagnosis of sinus disease was made and eventually substantiated in 9 cases of this series (cases 3, 5, 8, 9, 14, 15, 17, 18 and 22). I also wish to point out that of these 9 patients for whom a preoperative diagnosis of sinus thrombophlebitis was made, 6 failed to show at the time of the operation the usual external evidence that would lead one to suspect that the mastoids were sufficiently diseased to warrant surgical intervention. In all but 1 of the 9 cases in which a preoperative diagnosis was made a study of the blood revealed the presence of organisms before operation. Furthermore, in 6 of the patients the blood culture was positive in the first specimen. In other words, for a patient with otitis, chills and a septic temperature in whom a careful study excludes all other possible sources for the fever, it is deemed safe to venture a diagnosis of sinus thrombophlebitis. However, I do not wish to indicate by assuming such a position that with these findings alone one is to proceed immediately to ligate the jugular vein and obliterate the lateral sinus. It behooves the otologist to study the case

10. Greenfield, Samuel D.: Acute Mastoiditis, Lateral Sinus Thrombosis and Pyemia: Report of Case, with Operation and Recovery, Arch. Otolaryng. **3**:444 (May) 1926.

further, using all other means and methods available to establish a diagnosis. Only after collecting and correlating all the data at hand is he in a position to determine whether he should further observe the patient or whether the time has arrived when it is propitious to operate.

To illustrate the point in question, I wish to refer briefly to the case of a girl 11 years old in whom after acute otitis of two weeks' duration, chills and a septic temperature supervened. The otoscopic picture disclosed a resolving drum and no evidence that the mastoid was diseased. The temperature rose daily to 105 and even to 106 F. Repeated blood culture showed no organisms. The urine was carefully watched, and on the fifth day of the apparent sepsis a specimen revealed an abundance of pus cells. The source of the chills and fever was pyelitic and not otitic. The child recovered without operation.

Blood Culture.—A preoperative blood culture showed organisms in 19 cases of this series. In 14 the first specimen taken gave positive results, in 2, the second specimen; in 3 the third specimen, and in 1 the sixth specimen. Postoperatively, in 7 cases the first culture was negative; in 2 the second culture was negative; in 3 the third culture was negative; in 1 the fifth culture was negative; in 1 the tenth culture was negative; in 3 the cultures immediately after operation were negative, again became positive and finally became negative, and in 2 the postoperative cultures remained positive until death.

Of the 19 cases in which bacteremia was demonstrated, 17 showed Streptococcus haemolyticus (beta streptococcus, Brown) and 2 showed nonhemolytic streptococci (gamma streptococcus, Brown).

No otologist will minimize the value of demonstrating the presence or absence of bacteremia in otitic infection. It is the most conclusive evidence that the local otitic process has become generalized. It is certainly of paramount importance in evaluating the operative indications. I am of the opinion that in the absence of organisms in blood cultures from patients in whom during the mastoid operation actual visible disease of the sinus wall can be demonstrated in the form of thickening and granulations, exploration of the lateral sinus is contraindicated. According to Friesner,[11] the complication most feared in sinus thrombophlebitis is bacteremia. Therefore, if one fails to demonstrate its presence under the conditions outlined, the case remains one in which the expectant plan of treatment is to be continued. However, otologists are cognizant of two important facts: first, that otitic sepsis may exist without one's being able to demonstrate organisms in the blood stream, and second, that even a lateral sinus which appears to be normal may harbor a thrombus in its lumen. To illustrate the importance of waiting for a positive blood culture in certain cases before deciding to operate on the sinus, I wish to refer briefly to a case.

11. Friesner, I.: Personal communication to the author.

A boy [12] was admitted to the hospital on April 10, 1922, with a diagnosis of surgical mastoiditis on the left side. At the time of the mastoid operation a small area of exposed lateral sinus showed some fine granulations on its wall. After a week of low grade fever the patient had a chill followed by a rise in temperature to 104 F. A blood culture taken shortly after operation was reported as containing no organisms. The patient had rises in temperature on five successive days, not preceded by chills, but on two occasions followed by sweats. It would appear from the story that the indication was to explore the lateral sinus. This was not done, however, because the blood cultures remained persistently negative; the white cell and polymorphonuclear counts did not show any increase; the hemoglobin content remained well above 80 per cent, and the patient's general condition was good. On the sixth day after the initial rise the temperature went only to 101 F. and then became normal, after which the patient made an uneventful recovery.

It is in cases of this type especially, therefore, that the blood culture becomes a most important deciding factor.

Blood Count and Hemoglobin Content.—Leukocytosis in sinus thrombosis differs little from that observed in connection with other severe systemic infections. Ordinarily speaking, the leukocytosis measures the patient's efforts at combating the invading organisms. Thus, by further differentiating the type of white cells, V. Schilling [13] prognosticated as to the progress of the infection. The total white blood cell count in this series has shown rather wide variations. The lowest count obtained was 8,500 and the highest 29,900. The differential count varied from 74 to 92 per cent. It is apparent that little value can be attached to a single white cell count. However, if the count is repeated, some valuable information can be deduced. A rising white cell count and a proportionate increase of polymorphonuclears indicate that the infection is making headway. In a number of cases of this series when involvement of a sinus became apparent, successive counts showed a progressive increase in these elements.

It seems, on the other hand, that a low white blood cell count in sinus thrombosis may signify one of two conditions: first, it may indicate poor resistance in the defensive mechanism; second, it may point to the fact that the process has become well localized and under control. Thus, in cases 14 and 19 the total white cell count was 11,000 and 8,800, respectively. Both cases terminated fatally. In case 13 there was a white cell count of 8,500 despite the fact that the patient had the most extensive involvement observed in the entire series. The process, however, was well localized, and she recovered. Similarly, a progressive diminution in the number of red cells and the hemoglobin

12. Greenfield, Samuel D., and Friedman, J.: Three Cases of Mastoiditis with Interesting Complications, M. J. & Rec. **121**:278 (March 4) 1925.

13. Schilling, V.: Das Blutbild und seine klinische Verwertung, ed. 2, Jena, Gustav Fischer, 1926.

content speaks for the presence of a severe systemic infection. Taken in conjunction with the other signs and symptoms, it aids materially in arriving at a diagnosis in the more doubtful cases.

Changes in the Fundi.—Changes in the fundi are of great significance in connection with sinus thrombosis. This is especially true when these changes go beyond mere fulness of the retinal veins. Blurring of the disk margins and elevation of the nerve head may be detected even before the more pathognomonic signs of sinus thrombosis are in evidence. On the other hand, changes of the fundus may first make their appearance only after ligation of the jugular vein and after drainage from the lateral sinus has been established.

The changes of the fundus as a rule show a greater degree of involvement on the side which corresponds to the diseased sinus. I have never observed involvement of the fundus on the opposite side alone.

In this series changes of the fundus occurred in 8 patients, making a total of 36 per cent. Of these, 2 showed fulness of the retinal veins on the affected side; 3 disclosed blurring of the margins of the disk on both sides, and in 3 there was elevation of the nerve head. In 1 case the changes in the fundus appeared after ligation of the jugular vein. In all cases the fundus returned to normal within three to nine weeks.

DIAGNOSTIC IMPORTANCE OF THE PATHOLOGIC CHANGES OF THE SINUS WALL DURING THE MASTOID OPERATION

A diseased sinus wall in a patient with no symptoms to indicate the presence of sepsis calls for wide exposure of the diseased portion of the sinus wall. This should be carried out whenever possible to the extent of visualizing a circumference of normal sinus about the infected area. If there have been symptoms prior to the mastoidectomy that would lead one to suspect the possibility of involvement of the sinus although no blood culture has been taken before operation, the same procedure should be carried out. The patient is then entitled to further study and should be treated expectantly for the time being. I cannot agree with those who advocate the obliteration of the lateral sinus in every case in which extensive periphlebitis is found at operation, regardless of the presence or absence of symptoms pointing to systemic involvement. If one were to adhere to this principle, it must be apparent that many persons would fall into this group who under ordinary conditions get well without being subjected to this drastic procedure. In a routine mastoid operation I have sometimes unexpectedly come on a diseased sinus in which the periphlebitis was seemingly extensive. These patients presented neither signs nor symptoms that would lead one to suspect the presence of such involvement. Yet after exposure

of the diseased sinus wall as described, these patients have gone on to an uneventful recovery. Furthermore, even in cases in which the periphlebitis was accompanied by symptoms suggestive of possible sepsis, removal of all diseased bone and wide exposure of the sinus have caused an abeyance of symptoms in a number of instances, with ultimate recovery. However, I do not wish to give the impression that in all such cases the outcome has been so fortunate. I simply wish to record the fact that the line of differentiation in cases of this kind must of necessity be based on more substantial indications than the mere discovery of periphlebitis at the primary mastoid operation. It is only when organisms can be demonstrated in blood cultures or the symptoms of sepsis persist that surgical intervention is indicated.

This has been a general line of reasoning and the method employed in dealing with this series of cases. In no instance was the sinus obliterated without a positive blood culture, with the exception of cases 1 and 9.

In case 1 the diagnosis was made at the primary mastoid operation, at which time a necrotic sinus was found. The latter presented a fistula, from which free pus discharged. In case 9, the mastoiditis did not appear urgent, but the child had four rises in temperature preceded by chills. She had a white cell count that went from 19,000 to 26,000 cells. The hemoglobin content dropped from 65 per cent to 55 per cent, and her general appearance was one of sepsis. Two blood cultures taken on successive days were reported to be negative. Most important of all, a careful study failed to reveal any other source that would satisfactorily explain the septic picture. At operation a necrotic sinus with an organized thrombus in its lumen was found.

Cases 2, 4, 6, 7, 10, 12, 13, 16, 19 and 21 were all so-called postoperative cases, because the symptoms of sinus involvement became manifest some time after the original operation on the mastoid. Although at that time disease of the sinus wall was noted, exploration of the latter was deferred either until the blood culture became positive or until the symptoms of sepsis persisted. That undue delay was caused by waiting for a positive blood culture can easily be discounted by the fact that in the great majority of cases, as has been previously pointed out, either the first or the second blood culture contained organisms.

The condition assumes a much more complicated aspect when one is dealing with patients in whom both mastoids are affected. If one sinus appears normal and the other distinctly diseased, I am inclined to suspect the latter site as the source of the sepsis. If both are diseased, the problem becomes much more difficult. Signs and symptoms that appear insignificant at other times may be of great value to the otologist. The date of onset of the otitic infection may be of some aid. The side on which the otitis is of longer duration is more likely to be the offend-

ing one. The extent and severity of destruction of the mastoid cells should be taken into consideration. The one in which the process is more advanced is more likely to be on the side of the sinus through which the infection has gained access to the general circulation. Tenderness along the jugular vein should be looked for. The presence of a mass of small glands in the neck over the region of the vein may be detected. The Tobey-Ayer test finds its greatest usefulness in these cases. Another procedure, which is used a great deal in the foreign clinics and which helped me considerably in one case, is exploratory puncture of the lateral sinus. This is done for two purposes: First, to culture the blood from the venous tributary closest to the original site of the infection. The blood culture taken directly from both sigmoid sinuses should demonstrate theoretically a greater number of organisms on the side on which the sinus is diseased. Second, to determine whether or not there is a thrombus in the vessel. If an obiterating thrombus is present, no blood will appear in the syringe. However, the presence of free blood does not preclude the possibility of a mural thrombus. True, the latter, according to some, would tend to render the test unreliable. If the syringe failed to show free blood in a case which presents the problem of determining the involved sinus, no otologist would minimize the great value of such a finding. The test is employed not so much to show whether there is free blood in the sinus as to demonstrate its absence. It is in this connection that the test has its greatest usefulness. If one has witnessed the ligation of a jugular vein on the uninvolved side in a patient with bilateral otitic disease, one will become exceedingly hesitant in operating before exhausting all available sources in an earnest effort to single out the offending sinus.

So much for cases in which there is demonstrable inflammation of the sinus wall. I now come to a group of cases in which there is bilateral disease in which the blood culture is positive, in which the sinus walls are apparently normal and in which there is nothing to indicate which sinus is affected. The reaction to the Tobey-Ayer test is indefinite; free blood is demonstrated in both sinuses, and the sinus puncture shows little difference in the number of colonies grown. I have had 3 such cases. They have not been included in this series because I could not definitely classify the condition as sinus thrombophlebitis. There was no question that they were instances of sepsis following otitic disease. Failing to show which sinus was the portal of entrance to the general circulation, or rather failing to prove that the sinuses were responsible for conveying the infection, I deemed it advisable to resort to general supportive treatment. The first of these

3 cases[14] I reported in an effort to prove that there were channels other than those that have heretofore been described through which organisms may gain access to the general circulation from the mastoid cavity. In this case sepsis and bacteremia followed acute suppurative otitis media. At operation I found a large abscess cavity in an old reinfected mastoid. The sinus plate appeared normal, and wide exposure of the lateral sinus failed to disclose any macroscopic evidence of disease of its walls. Nevertheless, without ligation of the jugular vein and obliteration of the lateral sinus this patient recovered. There is no doubt that there must have existed a more direct line of communication between the abscess cavity in the mastoid and the blood stream. Similarly, in the second case, recovery took place but only after the development of suppurative disease of the hip and the elbow. In the third case the patient went on for six weeks with a septic temperature and finally acquired pneumonia but ultimately recovered. In all these 3 cases the futility of any operative procedure must therefore seem self evident. Friesner,[15] in this connection, likewise expressed the opinion that the infection passes directly through preformed pathways, in the form of either dehiscences or blood vessels.

THE EMISSARY VEIN IN SINUS THROMBOPHLEBITIS

Little attention has been paid to the emissary vein in connection with sinus thrombophlebitis. Its involvement secondary to disease of the sinus must occur often. In those cases of sinus thrombosis in which incision of the sinus is not followed by bleeding, the emissary vein must certainly share in the thrombotic process. Ziegelman[16] reported 2 cases in which he found the emissary vein thrombosed in disease of the lateral sinus, and he advised ligation of this vessel to limit the spread of the infection.

The emissary vein varies greatly in size. According to Streit,[17] it may range from 3 to 10 mm. in diameter. This is therefore at times an appreciably large vessel. Its length in the mastoid process depends on the distance from the mastoid foramen, the point of exit of the vein and its place of entrance into the lateral sinus. This distance is

14. Greenfield, Samuel D.: Bacteremia of Otitic Origin: Routes of Infection to the Blood Stream, Laryngoscope **43**:646, 1933.

15. Friesner, I.: Infections of the Middle Ear: Acute Systemic Infections from the Ear, Arch. Otolaryng. **14**:257 (Sept.) 1931.

16. Ziegelman, Edward F.: Surgical Importance of the Mastoid Emissary Vein in Infected Lateral Sinus Thrombosis, Arch. Otolaryng. **18**:298 (Sept.) 1933.

17. Streit, H.: Ueber otologisch wichtige Anomalien der Hirnsinus, über accessorische Sinus und bedeutendere Venenverbindungen, Arch. f. Ohrenh. **58**: 161, 1903.

governed to a great extent by the presence or absence of suprasinus and postsinus cells. It is obvious, therefore, that the emissary vein may become primarily implicated from direct extension of the inflammatory process in the mastoid cells. There is no doubt that the emissary vein is the seat of infection and its circulation becomes obstructed in many more instances than otologists have been led to suppose. Griesinger [18] called attention to a local swelling over the region of the emissary vein in those cases in which the circulation in the vein was obstructed. Owing to the fact that the process remains essentially local, there have been few or no manifestations pointing to the presence of such a condition. On the other hand, one of two other courses may be followed in those instances in which the emissary vein is an appreciably large vessel and becomes primarily involved. First, it may give rise to systemic manifestations similar to those occasioned by thrombosis of the lateral sinus itself. Surely this must appear to be a logical inference. Second, the mastoid emissary vein may be the primary seat of the thrombophlebitis extending therefrom into the lateral sinus and ultimately giving rise to general septicemia.[19]

Case 2 was one in which thrombosis of the emissary vein was noted at the primary operation. Preoperatively, this patient had a septic temperature suggestive of a possible involvement of a large venous radicle. The lateral sinus itself appeared normal at the primary mastoid operation. The signs and symptoms of sepsis continued. Eight days later when the lateral sinus was explored, it presented a markedly diseased and thickened wall. The emissary vein was necrotic and came away en masse. This was not followed by bleeding.

There was no question, therefore, that the emissary vein was the primary seat of the infection and that the lateral sinus became involved directly as a result of extension from the emissary vein. I must agree with Ziegelman as to the advisability of tying off the emissary vein when it is so thrombosed in order to avoid the complication which ultimately developed in my patient.

RECURRENCE OF SEPSIS

In the final analysis, all the surgical procedures instituted, both for the control and for the cure of sepsis secondary to thrombophlebitis of the lateral sinus, would be insufficient to accomplish this end were it not for nature's protective mechanism inherent in the human anatomy. There is no doubt, particularly in the light of more recent anatomic studies, that the circulation in the lateral sinus has a wide and varied

18. Griesinger, W.: Arch. d. Heilk. **3**:33, 1862.

19. Greenfield, Samuel D., and Friedman, J.: Primary Thrombosis of the Mastoid Emissary Vein with Secondary Involvement of the Lateral Sinus, Laryngoscope **33**:347, 1923.

anastomosis with the general system. This is made even more striking as the result of the work of Ottenberg.[20] He was able to demonstrate the presence of a greater number of organisms in the blood taken from the jugular vein on the normal side as compared with that from the vein on the diseased side. This then would tend to demonstrate most conclusively that whatever surgical methods are employed serve only as the first step in the efforts to cope with the infection. Operations have as their primary purpose the establishment of adequate drainage and the prevention, if possible, of additional hordes of invading organisms from entering the blood stream. The latter is further enhanced by the formation of protective thrombi in the various venous tributaries to and from the original site of infection in the lateral sinus. Thrombi have a twofold purpose: first, they act as physical barriers sealing up wherever possible those pathways through which organisms may enter, and second, they contain at their proximal end the defensive forces of the body. It is here that the battle rages and the organisms are destroyed and ultimately extruded. As the proximal end of the thrombus undergoes liquefaction, the distal end continues to grow. From this it is apparent that efforts must be directed toward aiding nature in the construction of these thrombi. In the vessels that are inaccessible there is little that the surgeon can do to effect this end. In the lateral sinus, however, much can be done.

Beck[21] attempted to study the histologic changes that take place in the lateral sinus as a result of compression of the sinus with a gauze plug. He tried to apply his findings in this case to what actually takes place in an infected sinus. It is obvious that the two are not exactly analogous, and therefore his deductions cannot be entirely correct. Kopetzky[22] in his "Critique of Beck's Experiment," although he was not in accord with Beck's conclusions, has nevertheless drawn some valuable deductions from them. Beck noted the failure of a thrombus to form in that part of the sinus in which endophlebitis was present. The formation of thrombi seemed to take place only at the point where normal intima was found. Kopetzky used this as a basis to explain the reason why in some cases bleeding persists on removal of the plugs from an obliterated sinus after from five to seven days, when in most instances thrombosis has occurred by this time at the site of the plugs. He attributes this to the fact that the latter have not been placed sufficiently beyond the site which corresponds to the endo-

20. Ottenberg, R. H.: Differential Blood Cultures, J. A. M. A. **94**:1896 (June 14) 1930.

21. Beck, O.: Histo-pathologische Veränderungen am Sinus sigmoideus des Menschen hervorgerufen durch Kompression, Acta oto-laryng. **12**:228, 1928.

22. Kopetzky, Samuel J.: Acute and Chronic Otitis Media and Sinus Thrombosis, Arch. Otolaryng. **10**:302 (Sept.) 1929.

phlebitis. It is a well known fact that the external appearance of the sinus wall does not indicate necessarily the extent to which the corresponding intima is involved. One must therefore bear in mind the importance of this fact when obliterating the sinus; that is, one must place the plugs well beyond the involved intima so as to insure the formation not alone of a thrombus but of one that is sufficiently extensive distally to withstand whatever liquefactive process may take place proximally. If the formation of thrombi keeps pace with liquefaction, the body is able to withstand the assaults of the invading host. If, on the other hand, the liquefactive process is too rapid or the defensive forces cannot be mustered out quickly enough to repel the invasion, then the organisms break through the weakened barriers and again enter the general circulation, and there is a recurrence of the sepsis. This recurrence may be due to extension of the disease in the direction of the torcular Herophili of the bulbar ends. In cases in which the latter is the seat of the infection there is likely to be tenderness and swelling in the region below the mastoid wound extending downward into the neck over the area corresponding to the upper portion of the severed jugular vein. In case 11 the reinfection resulted in local cellulitis of the neck. Symptoms of sepsis reappeared, and the temperature again began to show wide fluctuations, although the blood culture remained negative. Pressure upward along the course of the jugular vein caused a discharge of pus from the bulbar end of the sinus. After insertion of a drain down into the bulb, the septic symptoms quickly abated and the patient recovered.

More often, however, the sepsis comes from extension in the direction of the torcular Herophili. I reported two such cases,[23] cases 6 and 8 of this series. In both instances there was wide exposure of the sinus at the primary operation and the plugs seemed to have been placed well beyond the areas corresponding to the diseased sinus wall. Subsequent to ligation of the jugular vein and obliteration of the lateral sinus the symptoms of sepsis subsided. The blood cultures became negative, and it appeared that the process was well in hand. However, symptoms of sepsis recurred, chills reappeared, the temperature fluctuated widely and blood cultures again showed the presence of organisms. Both patients were reoperated on, and on exposure of the sinus in the direction of the torcular the source of trouble was easily ascertained. That portion of the sinus which appeared unaffected at the primary operation now showed definite disease. It was evident from this that the liquefactive process had extended beyond the confines of the thrombus and organisms were again gaining access

23. Greenfield, Samuel D.: Lateral Sinus Thrombo-Phlebitis with Extension to the Torcular: Report of Two Cases; Operation with Recovery, Laryngoscope **43**:751, 1933.

to the circulation. In each instance the sinus was exposed as far as the torcular and new plugs were applied. Both patients ultimately recovered.

SPONTANEOUS RUPTURE OF THE LATERAL SINUS

Spontaneous rupture of the lateral sinus presupposes the existence of disease in the sinus. It is inconceivable that such a complication could occur in a normal sinus. The latter is exposed too often and the occurrence of rupture is too rare for an attempt to be made to associate both with the former as an important causative factor. A normal sinus on which injury has been inflicted heals rapidly. Most physicians have had this experience. Disease of the sinus wall, especially when all the layers are implicated, is likely to lead to weakening of the vessel, with ultimate perforation. It is surprising, however, when one considers the frequency of sinus thrombophlebitis that spontaneous rupture of the sinus is not encountered more often. In this series there were only 3 instances in which this complication occurred despite the fact that in many cases of this series the sinus wall was extensively diseased, even to the extent of the presence of a fistulous opening in the vessel. Yet spontaneous rupture did not take place. There must be something aside from the disease of the sinus wall which is the determining factor in those cases in which perforation occurs. The methods in which the wounds are treated subsequent to the mastoid operation can hardly explain satisfactorily the etiologic factor. For rupture to take place, all the layers of the sinus wall must of necessity be involved. When the intima alone remains unaffected, and the other layers undergo rapid and extensive disintegration, the support and protection to the circulation is afforded only by this thin and delicate membrane. If the process continues for any length of time, the intima suddenly gives way before the continued pressure of the blood stream and rupture takes place. Concomitant with the absence of endophlebitis there is failure on the part of the blood stream to construct thrombi, the formation of which is so essential in plugging the leak, so to speak. On the other hand, when the process is not so rapid and the layers of the sinus wall do not disintegrate en masse, especially when the intima shares in some part of the inflammatory changes, which is the usual occurrence, enough time elapses for thrombi to organize. So if the condition progresses and a rupture takes place, nature is already well prepared to meet this exigency. In cases 7, 19 and 20 this complication occurred on the sixth, ninth and third day, respectively. In case 7 the blood culture was positive before rupture, and in cases 19 and 20 it became positive only after the rupture. In none of these cases were thrombi demon-

strable. Smith [24] in his report of 3 cases of spontaneous rupture of the lateral sinus was able to show the gross pathologic changes in the sinus walls as the condition progressed. He also concurred in the observation that thrombi were not demonstrable in these patients, so that the absence of formation of thrombi is most likely an important determining factor in those cases in which rupture takes place spontaneously. In case 7 recovery eventually occurred after a most stormy convalescence. Patient 19 did not recover. In the latter instance the sinus was packed off only after the patient had bled considerably. The blood culture, which before rupture was negative, became positive, and the patient became profoundly septic. She died several days later. Patient 20 was more fortunate. The sinus ruptured at the first dressing. Bleeding was easily and quickly controlled. Several days later I was able to enter the sinus and obliterate it. The patient made an uneventful recovery.

PARALYSIS OF THE ABDUCENS NERVE

This is not a common symptom observed in connection with thrombophlebitis of the lateral sinus. Its advent in otitic disease is fairly common, especially in infections of the middle ear with or without mastoid disease, the so-called Gradenigo cases,[25] and in suppuration of the petrous pyramid.[26] In the former it is part of the triad and is looked for early in the disease. In infection of the petrous pyramid it may occur as a late complication, but it is not essential in establishing a diagnosis. In the two conditions mentioned the pathologic process is based on a well known anatomic basis. But its occurrence in connection with thrombophlebitis of the lateral sinus was not satisfactorily explained. It was not until recently that I [27] reported a case of bilateral paralysis of the abducens nerve which suggested to me a plausible and satisfactory explanation as to the probable pathologic picture in these cases. The involvement of the abducens nerve on the unaffected side could not possibly have occurred as a result of direct extension from the petrous apex of the diseased side. To begin with, the patient had none of the symptoms indicative of disease of the petrous pyramid.

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24. Smith, A. J.: Spontaneous Perforation of the Sigmoid Sinus Following the Mastoid Operation: Report of Three Cases, Arch. Otolaryng. **17**:43 (Jan.) 1933.

25. Greenfield, Samuel D.: Gradenigo's Syndrome, Arch. Otolaryng. **9**:171 (Feb.) 1929.

26. Greenfield, Samuel D.: Conservative Treatment of Petrositis: Report of Two Cases with Recovery Without Operation, Arch. Otolaryng. **20**:172 (Aug.) 1934.

27. Greenfield, Samuel D.: Etiology and Pathology of Paralysis of the Abducens Nerve Associated with Sinus Thrombophlebitis, Arch. Otolaryng. **19**:336 (March) 1934.

Furthermore, if this condition were present, in order for an infection in one pyramid to migrate over to involve the nerve on the other side, one must presuppose the existence of rather extensive basilar meningitis. There were at no time symptoms suggestive of such a complication. However, with the changes that this patient presented at operation—extensive thrombosis of the lateral sinus from the torcular through the bulb into the jugular vein to the clavicle—one must conclude that a similar condition was present in one or both petrosal sinuses. The purely passive edema of the dura at the base of the brain incidental to the extensive venous blockage plus that occasioned by the phlebitis of the petrosal sinuses not only resulted in involvement of the abducens on the affected side but also readily extended across the medial line to involve its fellow. A study of the structures of this region immediately discloses the close proximity of the abducens nerves to each other and their intimate relationship with the petrosal sinuses. It is reasonable to conclude that the onset of paralysis of the abducens nerve in a patient with an otitic infection which runs its course with symptoms suggestive of sepsis is almost pathognomonic of thrombosis of the lateral sinus. This becomes even more certain in the absence of indications of suppuration of the petrous pyramid. In this patient the palsy on the affected side made its appearance two days after the primary operation on the mastoid. Palsy of the abducens nerve on the other side followed within thirty-six hours. In both instances the paralysis occurred before ligation and excision of the jugular vein and obliteration of the lateral sinus. Soon after the second operation the external rectus muscle on the normal side showed signs of regained function. The involvement of both external rectus muscles cleared up completely within four weeks.

TREATMENT

It is not my intention to dwell at any length on the much debated phases that comprise the treatment of thrombophlebitis of the lateral sinus with the view of advancing any new theories regarding procedure or technic. Too much time has been consumed in otologic debate, and too many pages in otologic literature have been devoted to endless discussion of these moot questions. I simply wish to record the sentiment of a conservative school in otology, which is unwilling to make any wide departure from the time-honored, well tried orthodox methods of treatment, particularly when they have yielded results that are at least as good, if not better, than those obtained by otologists advocating radical changes in those methods of therapy. Otologists must agree that all cases of sinus thrombosis are not alike and that no one set of rules can be propounded which will serve as infallible guides in their management. Each case is a law unto itself and therefore merits indi-

vidual deliberation and judgment, even to the extent of varying well established therapeutic measures when the occasion dictates a change in their judicious application.

Fundamentally, the modern treatment of sinus thrombophlebitis has not varied much from that advocated by Zaufal [28] almost sixty years ago, when he advised ligation of the jugular vein and opening of the lateral sinus in the treatment of otogenic general sepsis. These principles are just as valid today and are now based on even more scientific and clinical experience than was available in those days.

It seems that most otologists agree that before any radical surgical procedures are instituted in the treatment of thrombophlebitis of the lateral sinus one should be reasonably assured of the diagnosis. This having been established, the original focus in the bone is first to be thoroughly and completely eradicated. In the handling of the sinus itself each individual surgeon has his own method, but whatever procedure is employed, fundamentally, it has as its ultimate aim the establishment of drainage from the lateral sinus. The latter is materially enhanced by removing as much as possible of the parietal wall of the vessel. In regard to the removal of thrombi from the infected lateral sinus, it appears that no great risk is entailed by their dislodgment, whereas on the other hand it may serve the purpose of removing infection which at some future time may act as a source of trouble. It is true that new thrombi begin to form almost immediately after removal. But those that grow after drainage has been established from the sinus are likely to harbor less infection than those that have been formed with the vessel closed. After the operation on the sinus has been completed, it is inadvisable at future dressings to search and probe for thrombi in an effort to remove them.

Up to this point there seems to be little controversy in regard to the treatment of thrombophlebitis of the lateral sinus. However, in some of the foreign clinics treatment is extremely conservative, notably in that of the Kommune Hospital of Copenhagen, Denmark, from which Mygind [29] reported a series of cases of sinus thrombosis in which no other treatment was instituted beyond the performance of a simple mastoidectomy. The lateral sinus was not opened, and the jugular vein was not ligated. Surprisingly, he recorded recovery in 71 per cent of cases. To me this is almost incredible. But in an analysis of his cases I find an enormous number of patients, 41 per cent, in whom there occurred severe pyemic symptoms and extensive metastases. That many of these patients ultimately recovered is not

28. Zaufal, E.: Versuche mit dem Nitze-Leiter'schen Endoskop zur Untersuchung des Ohres, der Nase und des Nasenrachenraumes, Prag. med. Wchnschr. **5**:516, 1880.

29. Mygind.[3]

doubted. One must not lose sight of the fact, however, that in the treatment of thrombosis of the lateral sinus one endeavors not alone to lessen mortality, but—what is equally important—to lessen morbidity. I doubt whether otologists in this country are prepared to subscribe to such ultraconservative methods of treatment.

Perhaps the most controversial subject in all the realm of therapy is the matter of the jugular vein. Here again, there is no doubt that recovery occurs without ligation or resection, but this alone is not a sufficiently valid reason to discredit the procedure. Those who have used it feel that this is a most important and effective remedial agent in the hands of the otologist. Under ordinary conditions the procedure consumes little time, and especially with local anesthesia is no added shock to the patient. When this factor is at all under consideration, it can be performed some time after or before obliteration of the sinus. As a matter of fact, some of the adherents of ligation advise obliteration of the lateral sinus and ligation of the jugular vein in all cases at two separate sittings to minimize this operative risk.

The internal jugular vein in the neck is the direct continuation of the lateral sinus in the cranium, and is therefore the main channel through which blood flows from the latter to the former. It is only when the lower end of the sigmoid sinus is blocked that the current becomes reversed. Infection is propagated in the direction of the current of blood much more rapidly than against the blood stream. Certainly, no one will dispute the fact that bacteria and dislodged emboli are more likely to enter the general circulation by way of the internal jugular vein than through any other venous tributary. Thus, by ligation one cuts off the main pathway through which infection may enter and at the same time, as Braun [30] put it: "We set the lower limit to the portion of the venous system which is cut out of the circulation by the sinus operation." It further serves to slow down the blood stream in those cases in which an occluding thrombus has not formed. By so doing, it enhances the formation of thrombi, which are so important in both limiting the spread of infection and aiding materially in its eradication.

Whatever objections are raised to ligation of the jugular vein seem to be founded purely on theoretical rather than on basic scientific principles. Otologists know that metastases occur in bacteremia of all kinds and that they follow in the course of the disease. One can hardly justify the comment that they are the result of ligation of the jugular vein. The complications that follow occur not because of ligation but rather in spite of its employment.

30. Braun, Alfred: Sinus Thrombophlebitis: Inflammatory Diseases of the Venous Sinuses of the Dura Mater, New York, Paul B. Hoeber, Inc., 1928.

The jugular vein was ligated in all the cases of this series, and in a number of instances the sudden cessation of septic symptoms immediately after ligation was so striking that one must be forced to conclude that these changes were brought about by the employment of this procedure. In only one instance was the jugular vein resected. This was in case 13, in which a thrombus was demonstrated in the lumen of the vessel.

SUMMARY

Twenty-two cases of thrombophlebitis of the lateral sinus have been reviewed and studied.

Special attention has been called to the advisability of conservatism until the diagnosis has been fully established.

The following points have received special consideration:

1. Diagnostic importance of the pathologic findings in the sinus wall during the operation on the mastoid.
2. Disease of the mastoid emissary vein in sinus thrombophlebitis.
3. Recurrence of sepsis in sinus thrombosis.
4. Spontaneous rupture of the lateral sinus in disease.
5. Paralysis of the abducens nerve occurring in connection with thrombophlebitis of the lateral sinus.

Ligation of the jugular vein is urged in all cases.

CONTRIBUTION TO SYMPTOMATOLOGY OF FOREIGN BODIES IN THE AIR PASSAGES

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I have made a comparison of statistics concerning the incidence of foreign bodies in the respiratory passages of patients in the old Russia, the Soviet Russia and Western Europe. The following series were used: (1) that of Preobraschensky,[1] up to 1890, (2) that of Albrecht,[2] from 1911 to 1921, and (3) that of Chevalier Jackson.[3] Preobraschensky reported 702 cases. The foreign bodies in 119 cases were beans; in 103, bones; in 4, watermelon seeds; in 4, pumpkin seeds, and in 3, seeds of other melons. In 16 per cent the bodies were metallic. There was only one instance of a sunflower seed. In Albrecht's series there was a percentage rate of 40 for metallic bodies, 12 for bones, 12 for sunflower seeds, 6 for watermelon seeds, 4 for pumpkin seeds and 25 for beans.

It is evident from these series that aspiration of vegetable foreign bodies is rare in Western Europe. One author only, Bélinoff [4] (Bulgaria), reported many instances of vegetable seeds. A high percentage of vegetable seeds as foreign bodies is reported from the United States of America. In a paper emanating from Jackson's clinic [5] there were, among 100 consecutive cases, 45 instances of a peanut as a foreign body, 15 of a watermelon seed and 17 of a corn kernel, while in the rest, 23, the foreign body was a shell, a bean or other vegetable matter.

In Russia, particularly among the children, the principal foreign body aspirated is the sunflower seed. It occurs as a foreign body in not

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1. Preobraschensky, S. S.: Ueber Fremdkörper in den Athmungswegen, Wien. Klin. **19**:191, 1893.

2. Albrecht: Die Fremdkörper der Trachea und der Bronchien, in Denker, A., and Kahler, O.: Handbuch der Hals-Nasen-Ohren-Heilkunde, Berlin, Julius Springer, 1926.

3. Jackson, Chevalier: Bronchoscopy and Esophagoscopy: A Manual of Peroral Endoscopy and Laryngeal Surgery, Philadelphia, W. B. Saunders Company, 1927.

4. Bélinoff, S.: Der Melonenkern als Fremdkörper der Luftwege, Monatschr. f. Ohrenh. **59**:1253, 1925.

5. McCrae, T.: The Clinical Features of Foreign Bodies in the Bronchi, Lancet **1**:735 (April 12); 787 (April 19); 838 (April 26) 1924; abstr., J. Laryng. & Otol. **39**:324 (June) 1924.

less than 50 to 60 per cent of the cases, and in the central Russian provinces and in Siberia up to 80 per cent. Watermelon seeds predominate in southern Russia. In the 98 cases which I have observed (Donetz basin), watermelon seeds were more frequent than sunflower seeds. Among 93 children of the ages up to 5 years, watermelon seeds were removed from 54 and sunflower seeds from 26. The two constitute more than 86 per cent of all foreign bodies in children. Pumpkin seeds were encountered in only 3 cases. The Russian authors have reported a rate of incidence in children of 85 to 100 per cent. In my series the incidence in children up to 5 years amounted to 95 per cent, and in children up to 3 years, to 67 per cent.

Analysis of the causes of such frequent aspiration of foreign bodies in children reveals that it is due, not to lack of care, but more to the fact that the parents are not familiar with the fundamentals of the hygiene of eating, which consist of not hurrying, talking, laughing or running around while eating. Histories frequently showed that a child 2 or 3 years of age was given a piece of a watermelon without the seeds removed from it, or an egg in a shell or an apple was placed in the hand, and he was allowed to eat while playing, running and laughing.

Jackson and Jackson [6] said: "Mother thinks that the safest place in the world for the baby is in her arms; and in certain respects this may be correct; yet the dangers herein referred to may lurk right there."

The tremendous development of children's nursery playgrounds and homes in which the children are taught principles of hygiene and in which the use of sunflower seeds and watermelon seeds is excluded has markedly influenced the incidence of aspiration of a foreign body. The mothers receive special instruction in consultation stations. The clinic at the Pirogoff Hospital has not recorded a single case of aspiration of a foreign body in a child in the past two or three years.

A comparison of localization shows that in Western Europe it is most frequently in the bronchi, particularly the right main bronchus (70 per cent in Albrecht's series), while in Russia the localization is most frequently in the trachea. The explanation offered is that the sunflower and watermelon seeds are smooth and do not swell, while beans, which constitute the chief foreign body in Western Europe, do swell in moisture, as was shown by Else Kuntze.[7] Because of these physical properties, sunflower seeds and watermelon seeds do not become arrested in the bronchi but are found most frequently in the trachea

6. Jackson, C., and Jackson, C. L.: Fishing in Food and Air Passages, Hygeia **11**:46 (Jan.) 1933.

7. Kuntze, Else: Ueber Lokalisation der Fremdkörper in den Bronchien, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **106**:58, 1920.

in a movable state. The capacity to float was more marked in watermelon seeds than in sunflower seeds in my experiments.[8]

In a few articles by Russian authors I have read reports of instances in which a watermelon seed was found in the main bronchus when bronchoscopy was performed. I am inclined to ascribe this to the fact that in those cases a general anesthetic had been administered. As the introduction of the bronchoscope caused no coughing reflex, the seed remained in the bronchus, not being arrested there, however. Had coughing been elicited, the seed would immediately have passed into the trachea.

When in the first years of my work in this field I administered chloroform before removing foreign bodies from the air passages, I gave the children only a few drops of the anesthetic—just enough to permit my cutting the soft tissues without disturbing the patient. In all the cases I observed, as soon as the trachea was opened the coughing which resulted was sufficient to dislodge the seed.

Since I began to do laryngoscopic treatment without anesthesia, the watermelon seeds without exception have promptly entered the bronchoscope. The arrest in the bronchus may be caused by instrumental manipulation in the course of a bronchoscopy.[9] The injured mucosa swells up, and the foreign body becomes arrested.

The diagnosis of such bodies is not difficult. They produce a characteristic loud sound during a paroxysm of cough, because the freely movable body impinges on the lower aspect of the vocal cords. With watermelon seeds, the sound is loud and is easily heard. With movable sunflower seeds, it is muffled but likewise can be heard from a distance. This is explained by the fact that the latter are not as movable as the watermelon seeds. The former have a limited sphere of movement and only exceptionally strike against the vocal cords. In a tracheotomy the watermelon seeds spring into the opening, while the sunflower seeds have to be removed instrumentally.

When the diagnosis of a foreign body and its localization are not aided by the characteristic loud sound, one must resort to percussion and auscultation of the lungs and to roentgenologic examination. The last is valuable in a small percentage of cases only, since the seeds do not cast a shadow. The roentgenologic diagnosis carefully worked out by Manges,[10] of Dr. Jackson's clinic, based on interpretations of secondary effects on the thoracic and abdominal viscera, was given little

8. Jarosslawsky, W.: Ueber die Fremdkörper der Luftwege, Zhur. ushn., nos. i gorl. bol. **8**:423, 1931.

9. Belinoff: Ueber die vereinfachten Methoden der Entfernung von Fremdkörpern aus den Luftwegen, Zhur. ushn., nos. i gorl. bol. **7**:569, 1930.

10. Manges, W. F.: Non-Opaque Foreign Bodies in the Air Passages: X-Ray Diagnosis and Localisation, Brit. J. Radiol. **31**:119, 1926.

application in Western Europe and in Russia. I believe that it rather than the introduction of a contrast medium is the method of the future. Jackson (1918) was the first to introduce pulverized bismuth into a bronchus. At the present time, however, Jackson recognizes only two indications for the use of the method: first, the need to differentiate parabronchial atrophied lymph nodes from a foreign body, and, second, suspicion of perforation of the bronchial wall by a foreign body. Roentgenologic diagnosis of a nonopaque body, so far as it affects the thoracic and abdominal viscera, was well worked out by the Leningrad Roentgenologic Institute.[11]

The clinical picture accompanying the aspiration of these two foreign bodies, the watermelon seed and the sunflower seed, produces the following characteristic symptoms on the part of respiration and reaction of the pulmonary tissue: Respiration is little affected in the case of a watermelon seed, as has already been pointed out, by Bélinoff in 1925 and by me in 1931. Symptoms of suffocation are much more frequent in the case of sunflower seeds. This is due to the greater size, as well as to the fact that frequently the seed becomes arrested in the trachea or the bronchus. The lungs show little alteration in the case of the watermelon seed. In most of my cases I found an insignificant tracheobronchitis; in many cases, a few râles, and in only 1 case of 54, pneumonia. This is explained by the fact that the pulmonary ventilation is not interfered with, because the seeds do not become arrested and because little infectious material is introduced by their smooth slippery surfaces. Sunflower seeds have frequently caused pneumonia, bronchopneumonia and severe tracheobronchitis.

Seasonal incidence of aspiration of foreign bodies is characteristic in Russia. From August, the number of cases increases. In 84 per cent of my cases the aspiration has occurred between August and November, and the same is reported by all the Russian authors.

Tracheotomy was formerly the principal method in Russia, in some cases followed by laryngoscopy. This was due, to a great extent, to a lack of laryngoscopists and bronchoscopists, as well as to the limited number of bronchoscopes. Removal of foreign bodies was done in most instances by the general surgeon. This situation has markedly changed since the October revolution. The tracheotomy, however, is still the most frequently used method for the removal of foreign bodies. It is because of their wide experience with the latter method that the Soviet laryngologists introduced the method of primary suture of the tracheotomy wound after the extraction. I did not see any mention of it in the

11. Reinberg, S. A., and Simonson, S. G.: Veränderung der Lunge bei Verstopfung des Bronchialbaumes durch Fremdkörper und ihre röntgendiagnostische Bedeutung, Vestnik roentgenol. i radiol. **10**:95, 1932.

European or American literature. It is true, there are opponents of the method who point to the danger of submucous emphysema, passage of blood from the wound into the trachea or interference with breathing because of secondary swelling of the tracheal mucosa at the seat of incision. Experience shows that these hazards can be avoided by suturing the cartilage as well as the soft tissues and by leaving the lower angle of the incision in the skin unsutured. The fear of respiratory difficulties is justified when swelling of the tracheal mucosa is noted during the tracheotomy after the mucosa has been traumatized in the course of the removal of the foreign body; primary suture is contraindicated in such cases. I have used the primary suture method since 1928, and I have not observed in a single instance any complication arising from it. The primary suture has some advantages over the cannula method, such as shorter course, absence of a protracted, tiresome and nerve-racking handling of the cannula, absence of injurious effects on the pulmonary tissue from breathing through a cannula and the parents' objection to the cannula.

Tichomirow [12] reported, in 1931, 18 cases of removal of foreign bodies in young children with the aid of the Seiffert apparatus. Immediately after Tichomirow had made his report, Dawydow [13] reported his cases. In 1932, the same author [14] reported further simplification of the method with a Brüning speculum, which he had modified. My associates and I began to use Tichomirow's speculum in 1932 and were able to remove not only the floating bodies in the upper segment of the trachea but bodies arrested in the depth of the trachea as well. Since the introduction of Tichomirow's speculum, we have discarded the use of the bronchoscope and tracheotomy for the removal of foreign bodies in children. The technic of introduction of the speculum is simple, and the removal of the foreign body can be accomplished in every case without general anesthesia. The principal advantage of the method for younger children is the complete absence of reaction below the glottis.

The method of direct laryngoscopy for the removal of foreign bodies from the tracheas of younger children finds an ever increasing application in Russia.

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12. Tichomirow: Entfernung von Fremdkörpern aus den Luftwegen im Kindesalter mit Hilfe der Stützlaryngoskopie nach Seiffert, Zhur. ushn., nos. i gorl. bol. **8**:735, 1931.

13. Dawydow, I. E.: Zur unblutigen Entfernung von Fremdkörpern aus der Trachea im Kindesalter, Monatschr. f. Ohrenh. **68**:936, 1934.

14. Tichomirow: Eine weitere Vereinfachung der Methode der Extraktion von Fremdkörpern aus der Trachea, Zhur. ushn., nos. i gorl. bol. **9**:313, 1932.

ESOPHAGOBRONCHIAL FISTULA

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AND
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ROCHESTER, MINN.

Esophagobronchial fistula is a relatively infrequent anomaly or accident associated with a distressing train of symptoms. In the great majority of cases life expectancy is short and death ensues as a result of aspiration pneumonia, pulmonary abscess or gangrene. Many conditions may give rise to this distressing anomaly, but carcinoma, especially of the esophagus, tuberculosis and developmental defects are by far the most common causes. Vinson and Sutherland,[1] Pape [2] and Lebsche [3] have called attention to the fact that patients live for many years with broncho-esophageal fistula and survive recurrent attacks of pulmonary suppuration. Because of the comparative rarity of this complication, it is deemed worth while to report two cases in which the patient survived for many years.

REPORT OF CASES

CASE 1.—A woman aged 31 had enjoyed excellent health until eight years before her examination at the Mayo Clinic, when she first had noticed that after eating she might cough up small particles of food. The episodes had been associated with violent paroxysms of coughing and had continued for four years, in which time she had had an attack of pneumonia. Esophagoscopy had revealed an esophagobronchial fistula between the lower portion of the esophagus and the bronchus which passed to the lower lobe of the right lung. Attempts had been made to close the fistula by topical applications through the esophagoscope, and some slight improvement had occurred. The year before the patient came to the clinic, pneumothorax had been performed, and an incomplete collapse of the right lung had been done. This had been continued for approximately ten months, and definite improvement had occurred, especially in the prevention of pulmonary hemorrhage, which previously had been an annoying factor.

Examination at the clinic revealed a tall woman, who apparently was underweight; her weight had gone from 160 to 130 pounds (72.6 to 59 Kg.) in the preceding year. The value for the systolic blood pressure was 112 mm. of mercury, and that for the diastolic pressure was 80 mm. The pulse rate was 90

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1. Vinson, P. P., and Sutherland, C. G.: Esophagobronchial Fistula Resulting from Actinomycosis, Radiology **6**:63-64 (Jan.) 1926.

2. Pape, Rudolf: Eine chronische Oesophagus-Bronchusfistel bei Oesophagusdivertikel, Wien. klin. Wchnschr. **47**:1320-1322 (Nov. 2) 1934.

3. Lebsche, Max: Zur Pathologie und Klinik der Speiseröhren-Lungenfisteln, Deutsche Ztschr. f. Chir. **220**:249-257, 1929.

beats per minute, and the temperature was normal. Physical examination of the thorax revealed hyperresonance over the right side and absence of breath sounds. The roentgenogram of the thorax showed a diverticulum of the lower third of the esophagus, and complete collapse of the right lung; the mediastinal structures were displaced to the left. The concentration of hemoglobin in the blood was 71 per cent; the erythrocytes numbered 4,450,000 and the leukocytes 8,000 per cubic millimeter of blood.

Bronchoscopic examination with the area under local anesthesia revealed a small opening on the medial wall of the bronchus which passed to the lower lobe of the right lung. A small amount of barium which was mixed with blood was exuding from the opening. Pus was likewise coming from the bronchi of the lower lobe of the right lung. Two days later esophagoscopy revealed a small diverticulum which opened off the right wall of the lower portion of the esophagus. At the tip of the comparatively small diverticulum was a fistula which measured

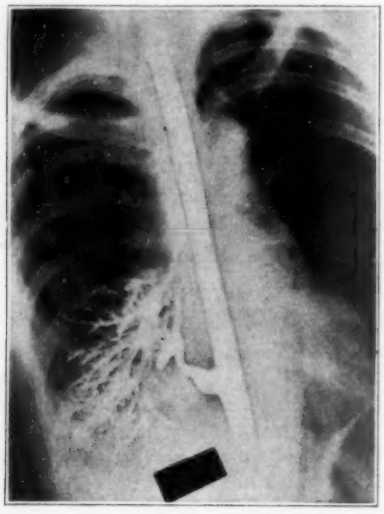

Fig 1.—Esophagobronchial fistula; the roentgenogram has been retouched to illustrate the appearance of the fistula after the injection of barium sulfate.

approximately ⅛ inch (0.3 cm.) in diameter (fig. 1). An aspirating tube could readily be passed into this fistula. Because of the recurrent attacks of hemoptysis and the annoying cough which occurred after eating, the collapsed lung was permitted to reexpand, after which the fistulous tract between the esophagus and the bronchus could be easily demonstrated by roentgenoscopic examination of the esophagus after a barium sulfate meal.

At operation the findings just described were confirmed. Tissue removed from the operative site failed to show any evidence of tuberculosis. The patient unfortunately died as a result of bronchopneumonia which involved the left lung. At necropsy, no further light was thrown on the exact etiologic basis of the condition.

Case 2.—This case is of special interest because of the long duration of symptoms and their comparatively mild nature. A man aged 52 came to the clinic because of attacks of strangulation and dyspnea which followed the sipping of

liquids. As a boy in Russia he had had similar attacks when he sipped tea, and because of such spells he had almost completely avoided drinking liquids. Ingestion of solid food seldom had caused trouble. He did not recall that he had expectorated solid food. At times, after eating, he had been annoyed by a peculiar sense of distress under the lower portion of the sternum, which had not occurred as a result of exertion. There had been a history of four attacks of pneumonia of the left lung.

The patient was a well developed, high-strung person who had a peculiar habit of stretching his neck and swallowing. His weight was 175 pounds (79.4 Kg.); the value for the systolic blood pressure was 104 mm. of mercury and that for the diastolic pressure was 80 mm. The pulse rate was 80 beats per minute, and the temperature was 98 F. The only physical finding of note was a few coarse râles over the base of the left lung posteriorly. Roentgenographic examination of the esophagus revealed an irregular fistulous tract, which extended

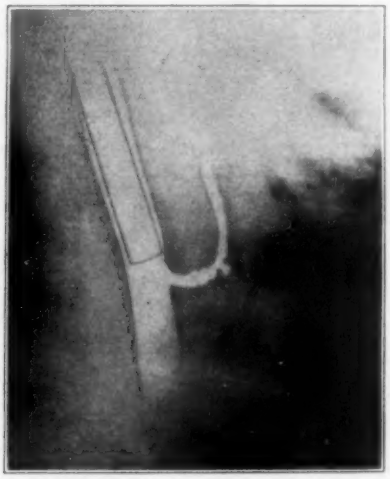

Fig. 2.—Esophagobronchial fistula; the roentgenogram has been retouched.

from the left lateral aspect of the lower portion of the esophagus and communicated with the bronchus of the lower lobe of the left lung. There was evidence of pneumonitis at the base of the left lung (fig. 2). The results of the laboratory examinations were essentially negative.

Esophagoscopic examination revealed a small fistula which opened directly into the lumen of the esophagus. The diameter of the opening was about ⅛ inch (0.3 cm). The fistula could easily have been overlooked, as it opened and closed rhythmically and was not visible when closed. There was no evidence of an associated diverticulum. A catheter could be readily passed into the fistula and into the bronchus. Iodized poppy-seed oil injected through the catheter was later coughed out by the patient.

It is interesting to speculate as to the possible etiology of the fistulous communication between the bronchus and the esophagus in these two cases. Carcinoma is the most common cause of this condition. The possibility of such a factor in these cases is not likely, in view of the

histories. Tuberculosis has been regarded as the most frequent cause of esophagobronchial fistula among adults; it causes traction diverticulums by scarring and contracture of old healing and calcifying tuberculous lymph nodes. This theory seems most tenable, especially if there is an associated diverticulum and in those cases in which the initial symptom does not occur before the third decade of life, as a traction diverticulum usually does not appear before this period. The condition in the first case that we have reported would seem to belong in this category. Ribbert, according to Lebsche, has postulated the theory that many of these fistulas which occur early in life and are of long duration result from developmental anomalies. Ordinarily, when one thinks of such an anomaly one thinks of one that is present at birth, which permits the patient to survive no longer than from three to four weeks. In the cases under consideration the fistula did not produce symptoms so early and the communication between the esophagus and the bronchus was so small that food or liquid did not readily pass into the bronchus. Because of the low degree of seepage and the duration of the infection, the bronchial mucosa apparently must undergo metaplasia, so that the mucosa can tolerate the infected material. Further emphasis is given to this theory since no postmortem evidence of tuberculosis can be discerned in such cases. The condition in case 2 would seem to fit into this category.

INTRADURAL ABSCESS COMPLICATING ACUTE MASTOIDITIS WITH SUBPERIOSTEAL ABSCESS IN AN INFANT

REPORT OF A CASE

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The following case report is submitted because of several unusual features which marked the course of suppuration of the temporal bone.

L. H., a baby 13 months of age, was referred to me by Dr. L. R. Willson on Jan. 8, 1937. The chief complaint as given by the mother was of a swelling behind the right ear. The familial and past medical histories were irrelevant to the present illness. On Dec. 26, 1936, an acute infection of the upper respiratory tract developed, as evidenced by nasal discharge and obstruction. The cold appeared to be gradually improving until Jan. 1, 1937, at which time the baby was irritable, felt feverish and would not take his feedings well. The parents believed that teething difficulties accounted for the illness. Late on January 8 a swelling was noted behind the right ear.

Examination revealed an acutely ill baby with a rectal temperature of 103.8 F. The tonsils appeared normal, and there was only a slight inflammation of the pharynx. Both nasal chambers were congested, and they contained a small amount of mucopurulent exudate. The right external auditory canal was clean, and the posterosuperior wall was drooping. The tympanic membrane was inflamed, except for the posterior portion, which was bulging and blanched. No perforation was noted, and the normal landmarks were obscured. There was a postaural swelling over the mastoid process the size of a pigeon's egg, which had a doughy consistency. The left external auditory canal was clean, and the tympanic membrane was inflamed and bulging posteriorly. Paracenteses revealed thickened drum membranes and purulent exudate under considerable pressure. A diagnosis was made of bilateral acute suppurative otitis media, with mastoiditis and subperiosteal abscess on the right side.

A complete mastoidectomy on the right side was performed on January 9, with the child under light ether anesthesia. The periosteal tissues were edematous, and several drachms of purulent exudate was observed coming from a fistula situated in the superior portion of the bony canal wall. On removal of the cortex empyema of the antrum and periantral cells was found. The tegmen antri was necrotic, and on its removal free pus was found between the bone and the dura. The dura was discolored and felt tense, but no granulation tissue was seen, such as is found in an epidural abscess. Sufficient bone was removed to expose healthy dura on all sides, and a discolored swelling one-half the size of a 10 cent piece was found on the dura. Near the anterior aspect a small fistulous opening was present, through which purulent exudate was pulsating. A probe could be passed into this opening less than 0.5 cm., and it was felt that the abscess was shallow. A crucial incision was made through the fistulous opening to permit more adequate

drainage, and the region was covered with a strip of iodoform gauze. The mastoidectomy wound was closed according to the usual technic, and the child left the operating room in good condition.

Because of the grave danger of the onset of generalized meningitis, immediate use was made of a derivative of sulfanilamide,[1] which is one of the chemotherapeutic agents recently developed and reputed to be of value in combating streptococcic infection. The derivative of sulfanilamide was given in 5 cc. doses every six hours for two days and then twice daily for the following three days. A tablet of sulfanilamide was given twice daily for twelve days. No local or general intolerance to the drug was noticed.

The postoperative course was essentially uneventful, except for a rectal temperature of 103.6 F. on the first postoperative day. The febrile reaction gradually subsided, and the temperature remained normal after the sixth postoperative day. At no time was there any suggestion of meningeal irritation, and the child played and ate normally from the day after operation. The packing was removed from the operative cavity on the fifth postoperative day. The external auditory canal was dry on the third day after operation and remained so thereafter. Drainage from the postaural incision continued until the eleventh postoperative day, at which time the edges of the skin became approximated; there has been no further discharge up to the time of writing.

The infection in the left ear began to subside on the fifth day of hospitalization, and by the tenth day the tympanic membrane had healed. The child was allowed to leave the hospital on the eleventh postoperative day.

COMMENT

This case was unusual for several reasons: First, the acute suppurative otitis media was undoubtedly of seven or eight days' duration without otorrhea. Second, it is uncommon to find so much destruction of bone in the tegmen antri of a baby having an associated subperiosteal abscess. Third, an intradural abscess being rare itself, it is unusual that the infection resolved without causing generalized meningitis or an abscess of the brain. (The lesion was classified as an intradural abscess because of the fact that there were no signs of extradural infection and its discreteness. In addition, the abscess cavity was small, with a smooth floor, and its propensity to resolution following adequate drainage further indicated its proper classification.)

1759 Fulton Street.

1. The derivative used was the disodium salt of 4'-sulfamidophenyl-2-azo-7-acetylamino-1-hydroxynaphthalene-3, 6-disulfonic acid, which is called "prontosil-soluble."

Clinical Notes

TONSIL DISSECTOR AND PILLAR RETRACTOR

THOMAS F. FURLONG JR., M.D., ARDMORE, PA.

With the aid of Mr. H. G. Herold of the George P. Pilling & Son Company, a tonsil dissector and pillar retractor was designed to incorporate into one instrument the salient features of two well known and much used instruments, the Hurd pillar retractor and the Freer elevator.

That part of the retractor which comes in contact with the anterior pillar and with which traction is exerted has had a semilunar curvature added to conform to the natural curvature of the pillar. This, I believe, permits a better exposure of the fossa and decreases the amount of trauma; consequently there is less discomfort to the patient when the instrument is used for examination in the office, in the

Fig. 1.—The tonsil dissector and pillar retractor.

performance of the tonsillectomy, in postoperative hemorrhagic measures or for routine postoperative examination. The handle is round and corrugated to provide a firm grip. This permits increased traction if necessary. The spatulated end of the instrument, to which a curve of approximately 45 degrees has been added, has two uses: first, as a dull dissector to enable one to free the tonsil, and second, as an elevator during the performance of a submucous resection when an instrument with a curve greater than that of the Freer elevator is necessary to dissect around marked spurs, ridges or curvatures of the septum.

This instrument may be obtained from the George P. Pilling & Son Company, Philadelphia.

36 Parking Plaza.

Read before the Philadelphia Laryngological Society, April 7, 1936.

From the Departments of Otolaryngology of the Graduate School of Medicine of the University of Pennsylvania, Service of Dr. George M. Coates, and the Pennsylvania School for the Deaf, Philadelphia.

Correspondence

OSTEOMYELITIS OF THE PETROUS PYRAMID

To the Editor:—Dr. Myerson in an article entitled "A Proposed Operation for Osteomyelitis of the Inferior Aspect of the Petrous Pyramid" (ARCH. OTOLARYNG. **25**:373 [April] 1937) cited a case of mine, of which he said: "Pus drained from the nasopharynx for five years . . . ."

This is not in accord with the facts, and the impression that a lesion could or would be allowed to drain for so long a period should not be allowed to pass without correction.

This patient was seen in 1930; she had been sick for many weeks and had had a radical operation on the mastoid. A pharyngeal fistula drained for four weeks, and in spite of this drainage meningitis developed, with streptococci in the spinal fluid.

At operation an enormous area of necrosis on the middle fossa side of the apex was found. Drainage was established, and the patient recovered.

Roentgen films taken five years after the operation (this is where the five year period was mentioned) failed to show any difference in the two pyramids in spite of the large amount of bone that had been extruded through the pharynx.

This case is an argument for drainage by way of the middle fossa and not by any inferior or pharyngeal route.

ROBERT L. MOORHEAD, M.D., Brooklyn.

Abstracts from Current Literature

Ear

AUDIOMETRIC STUDIES ON SCHOOL CHILDREN. A. CIOCCO, Pub. Health Rep. **51**:1609 (Nov. 20) 1936.

Studies were made on 1,400 school children in Washington with the 2-A audiometer of the Western Electric Company. The results showed that the curves found for children may duplicate any of the curves found for adults. A conduction lesion is responsible in about two thirds of the cases for impairment of hearing which involves tones of the middle and low ranges. The incidence of loss in high tones increases with age and is higher in boys than in girls. Normal tympanic membranes and membranes with slight change were found in 96 per cent of the ears which had good hearing, in 87 per cent of those with loss of high tones, in 80 per cent of those with impairment of hearing for conversation and in 56 per cent of those with conduction deafness. Apparently there is no relation between the type and degree of impairment of hearing and the condition of the nose and tonsils as observed at the time of examination.

SANFORD, Chicago. [AM. J. DIS. CHILD.]

THE MANAGEMENT OF OTITIS MEDIA AND SINUSITIS COMPLICATING THE COMMON COLD. LIONEL COLLEDGE, Practitioner **136**:33 (Jan.) 1936.

The management of suppurative otitis media calls for careful clinical observation. The common complications of meningitis, extradural or pernicious abscess and phlebitis of the lateral sinus must always be kept in mind. Colledge points out that there are two forms of mastoiditis complicating acute suppurative otitis media. In one type the organism is a hemolytic streptococcus causing a thin discharge. The reaction is severe; localization usually does not take place, and septicemia is a real danger. Hence, early operation is indicated. In the second type of mastoiditis the discharge is usually a thick pus, the process is localized and operation after several days usually brings about good results.

Another complication of the common cold which is discussed is acute sinusitis. The author stresses the point that a minimum of surgical intervention is the best treatment of the patient. The danger of setting up acute spreading osteomyelitis in the facial or frontal regions is a real one. Several cases are reported to illustrate the discussion on acute sinusitis.

SAPPINGTON, San Francisco. [AM. J. DIS. CHILD.]

PATHOLOGIC ANATOMY OF THE EAR: EMPYEMA OF PETROSAL APEX. H. RICHTER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **140**:360, 1936.

Richter shows that the diversity of the clinical course of empyema of the petrosal apex is explained by the variety of the anatomic aspects. His studies were made on fourteen patients, six of whom recovered. He describes the histologic aspects and attempts a systematic classification because he wishes to emphasize the dangerous position of all patients with acute otitis media whenever the healing of the otitis is retarded. In such cases the otologist should think not only of the well known endocranial complications but also of the possibility of the insidious development of empyema in the petrosal apex. The author thinks that if the latter possibility is recognized by a larger number of otologists, the number of fatalities from empyema of the petrosal apex will be greatly reduced.

EDITOR'S ABSTRACT.

CLINICAL ASPECTS OF OSTEOPSATHYROSIS. E. RUTTIN, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **141**:43, 1936.

Ruttin states that in 1922 he reported five cases of osteopsathyrosis and that since then he has observed five others. He cites the ages at which hardness of hearing was first noted. He investigated this to learn whether in these cases the hardness of hearing begins earlier than in ordinary otosclerosis. In the majority of cases the onset occurred at about the same time as in ordinary otosclerosis. However, it appears that in osteopsathyrosis the otosclerosis progresses more rapidly to a severe degree. About the relationship of the time of the fractures to that of the onset of otosclerosis, the author says that the fractures took place earlier. Whereas the fragility of the bones is greatest during childhood and decreases with advancing years, the otosclerosis shows the opposite behavior in that it increases with advancing years. From the clinical standpoint, the three main symptoms of osteopsathyrosis (blue scleras, fragility of bones and hardness of hearing) are not equivalent. The blue scleras are apparently already present during the fetal stage and later remain about the same; the fragility of the bones likewise begins during the fetal stage and decreases gradually during the post-fetal period, but the otosclerosis, for which only a predisposition exists, does not begin until about the time of puberty.

The author emphasizes that although both osteogenesis imperfecta and osteopsathyrosis idiopathica have been regarded as constitutional anomalies of the mesenchyme and of all its derivatives, it is nevertheless necessary to make a clinical differentiation between these two conditions. Regarding the transmission of osteopsathyrosis, the author says that his earlier material indicated that a parent with all three anomalies may have offspring with all three or only one or two, but that a parent with only one or two anomalies does not have offspring with all three. In the newer material, however, it was found that even parents without any of the defects may have offspring with all three. In the case under consideration, the grandfather had had the defects.

EDITOR'S ABSTRACT.

P-AUDIOMETER. F. GROSSMANN and K. SPIELMANN, Monatschr. f. Ohrenh. **70**: 195 (Feb.) 1936.

Grossmann and Spielmann describe a new audiometer, the so-called P-audiometer. The instrument is built into a portable case, and its operation is independent of the current supplied by the electricity network. The production of the sounds is done according to the principle of superposition. Especial attention was given to the calibration of the intensity of the P-audiometer. For this purpose a new method was developed which permits the graphic recording of the acoustic frequency progress, which in the customary diagram is parametrically plotted as a group of curves. The group of curves is found by comparison with an absolutely calibrated sound emitter. The calibration was done by means of Raleigh's disk. The group of curves makes it possible to record the threshold curve, which is supplied by the apparatus, directly into an absolute diagram. The results are important for the testing of the capacity for hearing. The authors stress the advantage of the introduction of the band of intensity in which the threshold curve is located.

EDITOR'S ABSTRACT.

CALORIC TESTS OF THE LABYRINTH IN CASES OF TUMOR OF THE BRAIN. H. BRUNNER, Monatschr. f. Ohrenh. **70**:206 (Feb.) 1936.

In describing the results of the caloric tests in cases of cerebral tumor, Brunner disregards the tumors of the cerebropontile angle, which he discussed in earlier contributions, as well as those of the medulla oblongata, the pons and the hypophysis. The remaining tumors (eighty in all) he differentiates into infratentorial and supratentorial. The caloric tests revealed normal irritability as well as deviations from the normal. The deviations appeared more often in the

form of increased than reduced irritability. A complete lack of response was never observed before the operation either in subjects with infratentorial or in those with supratentorial tumor. Normal irritability was detected in about 30 per cent of the cases. In this connection the author points out that the number of cases of tumor of the brain, in which the caloric irritability is normal (30 per cent) corresponds largely to the number of cases without ocular symptoms but that, on other hand, a normal caloric irritability is less frequent with tumor of the brain than is a normal roentgenogram. The fact that the caloric test in a case of tumor of the brain may disclose reduced excitability as well as hyperirritability is a disadvantage; however, the author found that in case of metastases in the posterior cranial fossa the labyrinthine irritability is often reduced.

Further, the author gives his attention to the labyrinthine hyperirritability. He observed it in association with 56 per cent of the infratentorial and with 67 per cent of the supratentorial tumors. He says that in determining the cause of this hyperirritability it is necessary to differentiate between infratentorial and supratentorial tumor. He finds that in a case of infratentorial tumor, the labyrinthine hyperirritability is a sign of irritation of the nystagmus centers in the brain stem. It is produced by the local pressure of the tumor on the brain stem. In accordance with the unilaterality of the tumor or with the stronger development of the tumor on one side, it is generally more intense on one side than on the other. In cases in which the pressure is severe the centers producing nystagmus may pass from a stage of irritation into a stage of paralysis, and in accordance with this the hyperirritability may change into a hypofunction of the labyrinth and finally even into complete unresponsiveness. Investigations of the causes of the hyperirritability in cases of supratentorial tumor revealed that it is only partly a true labyrinthine hyperirritability. It is largely an oculomotor irritability produced by a lesion of the cortical visual centers and tracts. This oculomotor irritability is either unilateral (in the cold test generally contralateral to the focus) or (during the period of compensation) bilateral. There is no reversion of this hyperirritability into unresponsiveness.

EDITOR'S ABSTRACT.

METASTASES IN OTOGENIC PYEMIA OF CHILDHOOD. IGNAZ HOFER, Wien. klin. Wchnschr. **49**:42 (Jan. 10) 1936.

Hofer states that if in the course of otogenic pyemia the symptoms persist even after the primary foci of infection have been treated, then the possibility of metastases must be considered. If there is local pain, the diagnosis is not difficult. Pulmonary metastases, abscesses and infarcts can be confirmed by roentgenograms, although there may be few symptoms. This may be the case when the metastases do not reach the pleura and there is no pleurisy. The prognosis of suppurative metastases in the hip joints, psoas muscle and pelvis is especially bad. Suppurative metastases in the hip joint usually do not remain confined to the joint but cause the formation of pus in the capsular ligament or inflammation of the bursa, which lies between the joint and the ileopsoas muscle, which may lead to continuation of the suppurative process in the fascia of this muscle, in the muscle itself and in the pelvis. Operative drainage of the pelvis is often difficult. The pus is retained, and there are attacks of severe fever and often hemorrhage. The child is weakened, and death often results from the suppurative process.

The treatment of metastases is usually operative, especially when other means remain ineffective. Cutaneous and superficial muscular abscesses may be treated conservatively or incised if they do not disappear. Individual pulmonary abscesses are curable. They may be coughed up or operated on by costal resection, especially if they have penetrated the pleural cavities and caused suppurative pleurisy. Metastases of joints which are small and near the surface may be treated conservatively. Larger joints may have to be punctured and drained.

Hofer's experience indicates that the formation of pus in the hip joint should be treated early by operative measures. In one of his cases of metastasis of the hip joint, however, the suppurative process reached the pelvis in spite of drainage

and finally caused death. He expresses the belief that a more radical treatment might have been successful. In a second case there was spontaneous dislocation of the condyle, leading eventually to suppuration in the pelvis. Surgical removal of the necrosed bones and resection of the joint brought about a cure, although the bone was slightly shorter and the joint remained slightly stiff.

The author reports three other cases of metastasis. In each case there was localization of the process in only one part of the body: in the first patient, in the cerebrum; in the second, in the hip joint, and in the third, in the kidneys. The patient with metastatic abscess of the cerebrum could not be saved, although the abscess was incised. Operative treatment of the ear and of the hip joint saved the second child, and operation on the ear combined with internal treatment (intravenous injection and chemotherapy) saved the third.

ABT, Chicago. [AM. J. DIS. CHILD.]

OTO-AUDION TESTS FOR HARD OF HEARING AUTOMOBILE DRIVERS. G. STEINBERG, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:442, 1936.

Steinberg points out that for automobile drivers the hearing of signals is of primary importance and that consequently tests should be made chiefly of their capacity for hearing fones, since this is far more important for the hearing of signals than is the capacity for hearing spoken words. It is important to know within what sphere of sound the most common signals of vehicles fall. Inquiries convinced the author that sounds of a frequency of less than 250 cycles are of slight importance for automobile drivers. The upper limits he places between 4,000 and 5,000 cycles. The hearing in noisy surroundings is likewise important for automobile drivers, and their hearing capacity should therefore also be tested in this respect.

The author examined the hearing of applicants for automobile driver's licenses by means of the oto-audion, giving attention to both ears and thus obtaining the so-called binaural oto-audion curve. He found that the oto-audion alone will generally decide what degree of hardness of hearing will permit safe driving. To be sure, for a certain degree of hardness of hearing the oto-audion test is not enough and test drives are necessary to determine the hearing capacity. Such test drives reveal that one may be a safe driver with a hearing capacity of 70 per cent of normal. The author emphasizes that this 70 per cent normal capacity refers not to the hearing capacity for the speaking voice, but rather to the hearing capacity for signals. The signals of vehicles are so high above the threshold values that they can be heard by persons with greatly impaired hearing. As another advantage of the oto-audion curve the author stresses its value in after examination of persons with progressing deafness in that it permits a comparison of successively taken curves.

EDITOR'S ABSTRACT.

Pharynx

REMOVAL OF TONSILS BY ELECTRICAL CURRENTS OF HIGH FREQUENCY. ALBERT EIDINOW, Brit. M. J. **1**:152 (Jan. 25) 1936.

Eidinow's conclusions are as follows: "The application of high-frequency electrical currents, commonly called surgical diathermy, may be employed successfully to remove and destroy the diseased tonsil. The technique of progressive destruction by coagulation has advantages, since this operation can be performed while the patient continues with routine duties. The procedure is slow, involving treatment at weekly sessions for six to eight weeks. The discomfort and disturbance incurred by the patient is not really great, especially in suitable types of cases, but in the highly strung and nervous patient it causes apprehension and fear. However, I have managed to complete treatment in all the cases I have attempted, and no patient has, as yet, failed to complete treatment once he has agreed to start."

ROYSTER, University, Va. [AM. J. DIS. CHILD.]

TREATMENT OF ACUTE TONSILLITIS WITH ANTIDIPHTHERITIC SERUM. M. SCHACTER, Med. de los niños **37**:33 (Feb.) 1936.

When acute tonsillitis assumes epidemic proportions, diphtheria should be suspected and prophylactic serum used. Schacter gives one dose of 5,000 units of antitoxin to patients with acute tonsillitis with high temperature, dysphagia and the usual bacterial flora. Even when the tonsillitis is not due to Loeffler's bacillus, the condition seems to improve rapidly.

DWYER, Kansas City. [AM. J. DIS. CHILD.]

CLINICAL SYMPTOMS OF CHRONIC TONSILLITIS. Z. WEIN, Monatschr. f. Ohrenh. **70**:310 (March) 1936.

Wein discusses the characteristics that determine the diagnosis of chronic tonsillitis and are important in deciding the advisability of tonsillectomy. First, he gives his attention to the perfusion with blood and the thickness of the mucous membrane covering the tonsils. If the tonsils are inflamed, the mucous membrane covering them likewise exhibits inflammatory changes (thickening and swelling). However, care is necessary, for the inflammation of the tonsillar mucous membrane not only may be caused by an inflammatory process in the tonsils but also may be a sequel of pharyngeal catarrh. The author emphasizes that not all cervical pains, difficulties in swallowing or sensations of foreign body are caused by the tonsils.

Further, Wein gives his attention to the hypertrophy of the tonsils, pointing out that swelling and enlargement are usually a sign of chronic inflammation; in rare cases, however, a hidden chronic abscess may be the cause of circumscribed protrusion of the tonsils.

The investigation of the firmness of the tonsils is likewise important. In diseased tonsils hardening of the whole or of parts is perceptible. Sensitivity to pressure deserves attention, particularly when there is a difference between the two sides. In discussing the importance of the test for expression the author points out that expression of detritus from the supratonsillar fossa does not necessarily indicate chronic tonsillitis, but if detritus and particularly pus can be expressed from the numerous lacunae, the presence of inflammation may be assumed. The appearance of fluid pus on slight pressure is the surest sign of chronic tonsillitis, but if infiltrated pus shines through the superficial follicles, this likewise is a sign of chronic tonsillitis. Fixation of the tonsils by peritonsillar cicatrization and adhesions is another sign of chronic tonsillitis, and the author thinks that this sign is not given sufficient attention.

Further, he discusses the inflammation of the regional lymph glands. He admits that cervical lymphadenitis may be caused by other factors than chronic tonsillitis; however, in many cases it is indicative of chronic tonsillitis, particularly if the inflammatory symptoms of the tonsil and of the lymphatic apparatus are homolateral. He advises that in the examination of the tonsils the palpation of the cervical lymph glands should not be neglected.

In the last part of his paper he stresses the importance of the anamnesis, pointing out that in order to determine the advisability of tonsillectomy, the symptom of chronic tonsillitis should be evaluated in the light of the anamnesis.

EDITOR'S ABSTRACT.

Larynx

A CASE OF AN IGNORED FOREIGN BODY IN THE TRACHEA. SUNE MEDAN, Paidoterapia **13**:793 (Oct.) 1934.

An instance of undiagnosed foreign body in the trachea of an 18 month old infant is reported. Because of the impression that the infant had pneumonia, the foreign body was overlooked on the first roentgenograms, which when later

reviewed revealed its presence. The case proves the great advantage of direct laryngoscopic examination in children.

HERRON, Spokane, Wash. [AM. J. DIS. CHILD.]

SCIENTIFIC PRINCIPLES OF THE PROBLEM OF REGISTER. D. WEISS, Monatschr. f. Ohrenh. **70**:562 (May) 1936.

Weiss points out that the term register should really be discontinued and that instead of talking of head and chest registers, the terms head and chest tones or head and chest tone functions should be employed. Nevertheless, for the purpose of the present discussion, he still adheres to the term register. He emphasizes that the vocal organ is endowed by nature with the capacity of an entirely uniform formation of voice in that the change of the different voice-forming components takes place gradually and without break. That this gradual transition is interfered with at certain points and results in "register" manifestations is a pathologic process. The direct cause of this process is sudden changes in the form, tension and function of the vocal cords and retro-active manifestations of the vocal tube. A physiologic manner of phonation will counteract these manifestations.

EDITOR'S ABSTRACT.

TUBERCULOSIS OF THE THYROID GLAND WITH TWOFOLD PERFORATION OF THE ESOPHAGUS. W. RUTHE, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:450, 1936.

Ruthe shows that although tuberculosis of the thyroid gland is not impossible, as was believed at one time, certain characteristics of the thyroid parenchyma provide considerable protection against tuberculosis. Nevertheless, in cases of miliary tuberculosis, nodules do appear in the thyroid. Regarding the case under consideration, the author says that the condition is a rarity because of the twofold perforation of the esophagus but that it is noteworthy chiefly on account of epithelial changes which were produced by the inflammatory process and which might lead eventually to diagnostic errors. This case demonstrates too that the clinical manifestations of tuberculosis of the thyroid gland are slight. The patient, a woman aged 59, was hospitalized on account of general exhaustion and constantly increased temperatures. A sudden and repeated hemoptysis, with a sensation of pressure in the gastric region, seemed to suggest carcinoma of the stomach with pulmonary metastases. The necropsy, however, revealed the unusual cause of the fatal hemoptysis, two perforations of the esophagus. Exposure of the right side of the neck revealed no thyroid tissue, but caseous foci surrounded by glassy tissue were seen. In the upper portion there was a large cavity, filled with bloody, smeary masses. The left lobe of the thyroid gland did not show the large caseous foci but consisted of tendinous, hard tissue. The histologic examination of this left lobe revealed atrophy and splintering of the thyroid tissue by chronic inflammatory processes. Moreover, there was a transformation of the epithelial tissues and they were grouped into nests resembling carcinoma. The author expresses the belief that this might easily lead to an erroneous diagnosis of carcinoma. He emphasizes that these epithelial changes in the thyroid gland are not characteristic for tuberculosis of this organ, for they are found in all chronic productive inflammations of the thyroid gland.

EDITOR'S ABSTRACT.

A SIMPLE TREATMENT OF CROUP. HERMAN VON WILLEBRAND, Finska läk.-sällsk. handl. **78**:605 (Oct. 11) 1935.

Von Willebrand has treated croup with suction of the membranes by means of an electric vacuum cleaner armed with a rubber tube and a Nélaton catheter. If necessary, the suction can be combined with intubation. Twenty patients have been treated with this method, of whom two with streptococcic laryngitis died.

WALLGREN, Göteborg, Sweden. [AM. J. DIS. CHILD.]

Nose

RHINORRHEA IN CHILDREN WITH SPECIAL REFERENCE TO THE POSTTONSILLECTOMIZED CHILD. ABRAM H. PERSKY, Arch. Pediat. **53**:1 (Jan.) 1936.

Persky suggests lines of approach for those children in whom infection of the upper respiratory tract does not clear up after the removal of adenoids or tonsils.

WADDELL, University, Va. [AM. J. DIS. CHILD.]

STUDIES ON THE COMMON COLD: VI. CULTIVATION OF THE VIRUS IN TISSUE MEDIUM. A. R. DOCHEZ, K. C. MILLS and YALE KNEELAND JR., J. Exper. Med. **63**:559 (April) 1936.

Studies of the cultivation of the virus of the common cold in a tissue medium and the capacity of the cultured virus to induce infection in human volunteers are reported.

Detailed descriptions are given of the methods employed to isolate, preserve and cultivate the virus and to test its activity in human volunteers.

The virus of the common cold can easily be isolated from properly selected patients and cultivated in a tissue medium.

When kept in the original nasopharyngeal washings, the virus will survive at icebox temperature under anaerobic conditions for at least thirteen days.

If the nasopharyngeal washings are frozen and dried in vacuo, the virus retains its activity for at least four months.

The virus of the common cold has been proved to multiply in a medium containing chick embryo tissue. Such cultures retain their capacity to produce typical infections in human beings for many transfers, involving several months. Attempts to cultivate the virus have been successful in seven of eight instances.

Prolonged cultivation of the virus in a tissue medium eventually leads to a loss of activity.

KNICKERBOCKER, Dallas, Texas. [AM. J. DIS. CHILD.]

PAUSTEREORAMA OF POLYPOUS MUCOUS MEMBRANE OF THE SINUSES. R. MITTERMAIER, Hals-, Nasen- u. Ohrenarzt (Teil 1) **27**:2 (March) 1936.

Mittermaier, although he is of the opinion that roentgenographic examination of the accessory sinuses can usually be done satisfactorily without the use of a contrast medium, admits that there are some cases in which a contrast medium is desirable. He regards as most suitable one with some adhesiveness to the mucous membrane; that is, the medium should outline the contour of the mucous membrane and yet leave the cavity of the sinus free, so that an air space remains. The author's attention was called to a contrast medium that fulfils these requirements. The substance is barium sulfate to which, for greater adhesiveness, tragacanth has been added. It is widely used in roentgenographic examination of the gastro-intestinal tract. The author tried it in the examination of the maxillary sinuses and found it highly satisfactory.

EDITOR'S ABSTRACT.

THERAPY OF OZENA. A. GLASSCHEIB, Monatschr. f. Ohrenh. **70**:166 (Feb.) 1936.

Glasscheib points out that avitaminosis plays a part in ozena, the deficiency involving chiefly the vitamins A and D. He cites experiments with animals in which an ozena-like condition was produced by means of a diet that was free from vitamins A and D. The avitaminosis results in dystrophic processes of the different nasal tissues (mucous membrane, bone and cartilage) and of the endocrine organs. Moreover, by the lack of vitamin A the resistance to bacterial infections is reduced and the deficiency of vitamin D results in alkalosis of the mucous membrane, which in turn results in bacillosis and suppurating secretion. The bacillosis and the casting off of cornified epithelial lamellae with pus cause the crusts and the fetor.

In accordance with this theory of the pathogenesis of ozena, Glasscheib suggests the following treatment: To stimulate the hypoplastic and atrophic glands, he vaccinates once a week with the cutivaccine of Paul or with a rheumatic cutivaccine,

and to increase the resistance to bacteria, he administers vitamin A by giving twice daily 5 drops of a vitamin A preparation and the same dose of a vitamin D preparation. The vitamin preparations can be replaced by medication with phosphorus and cod liver oil (twice daily a tablespoonful). To increase further the resistance to the saprophytes of ozena, the author gives once a week an intracutaneous injection of 0.2 cc. of his vaccine (autolysate of the bacteria Perez, Abel Löwenberg and pseudodiphtheria). Since the production of fever is contraindicated in ozena, the author emphasizes that the vaccination is made only intracutaneously. After from three to five vaccinations the crusts are cast off and the suppurating stage follows, which lasts from ten to forty weeks and which in turn is replaced by a mucous secretion, which finally leads to cure. The author describes the clinical histories of five patients who were treated with this method. In all he treated twelve cases, in eight of which the condition was cured, in two the fetor was lost and in the two others, the fetor and the crust formation were lessened.

EDITOR'S ABSTRACT.

THE EFFECT OF THE CALDWELL-LUC OPERATION ON THE TEETH. GERDA SCHMIDT, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:355, 1936.

Schmidt reports studies on forty-five patients with sixty-three surgically treated maxillary sinuses. In order to detect disturbances in the sensitivity of the teeth, d'arsonvalization was resorted to. Of the 425 teeth examined, 39 (10.8 per cent) showed permanent anesthesia and 2 were injured by the surgical intervention, so that the pulp became necrotic. The author observed that the disturbances in sensitivity which resulted from the radical Caldwell-Luc operation were limited to the premolars, whereas other investigators found the canine tooth and the first molar likewise involved. In discussing the importance of the desensitization of the teeth, the author emphasizes that desensitization is not synonymous with devitalization. The preservation of the blood supply is the only factor that is important for the life of the pulp, and since the abolishment of the sensitivity does not prove an interruption of the blood supply, the disturbance in the sensitivity that may result from the Caldwell-Luc operation is no contraindication against this method, the less so since the disturbances in sensitivity are usually only temporary.

The author states further that other investigators found more extensive disturbances following Denker's modification of the operation. Since Denker's method is only rarely employed at the author's clinic, she did not have the opportunity to verify this; however, she considers more extensive disturbances probable, in view of the removal of greater portions of bone in Denker's modification. She mentions authorities who prefer the operation of Caldwell and Luc for patients with well preserved teeth.

EDITOR'S ABSTRACT.

Miscellaneous

SPEECH DISTURBANCES IN CHILDREN AND THEIR CORRECTION. H. GUTZMANN, Gesundh. u. Erziehg. **48**:233 (Aug.) 1935.

Gutzmann discusses the various speech defects in children who have no anatomic deformities and no mental disturbance. These are stuttering, stammering, word deafness and disturbances of the voice. He stresses the importance of a complete history and thorough physical and mental examination, with an evaluation of the parental and environmental factors.

Psychotherapy and training are of importance. The methods already in use should be stopped; instead the parents should be given written instructions to follow. Special schools and classes are of value. Stammering is often due to organic changes. Thus, a thorough examination for defects should be undertaken. Word deafness may be difficult to identify, but one should suspect this when there is a delay in the time of talking. The use of a mirror is helpful in this type, but continued cooperation of the parents, instructor and child are essential. Dis-

turbance of the voice itself is most frequently caused by so-called voice nodules. Excess crying may be responsible, and it is seen more commonly in urban than in country communities. Certain infections, such as tuberculosis, may also cause hoarseness. Harmonic vibrators and electromechanical tone control together with local therapy may be useful in this type of disturbance. The author believes that the earlier these various types of disturbances are treated the better the prognosis.

HANSEN, Minneapolis. [AM. J. DIS. CHILD.]

CASE OF CARCINOMA OF BASE OF SKULL WITH BEHR'S SYNDROME. H. MELLER, Monatschr. f. Ohrenh. **70**:540 (May) 1936.

Meller directs attention to a syndrome which Behr described in 1925 and which is helpful in the localization of a malignant growth at the base of the skull. In the cranial portion of the pterygopalatine fossa, the maxillary nerve and the abducens nerve come close together. Consequently, these nerves become involved in case of the perforation of a tumor (usually of the sphenoid or maxillary sinus) into the pterygopalatine fossa.

Meller observed a patient in whom involvement of the maxillary nerve and the abducens nerve was the most outstanding symptom. An acute inflammatory intracranial process could be excluded, because there was no acute otitis. An intracranial tumor with distant action on the abducens nerve could be ruled out in view of the lack of other symptoms of tumor. The concurrence of paralysis of the abducens nerve with involvement of the second branch of the trigeminus nerve made it appear probable that the process was localized in the upper portion of the pterygopalatine fossa, and the roentgenogram corroborated this assumption.

Thus, it appears that the concurrence of paresis of the abducens nerve with anesthesia in the region that is innervated by the second branch of the trigeminus nerve (as was already pointed out by Behr) not only is an important symptom in the topical diagnosis of paresis of the abducens nerve but is indicative of processes in the pterygopalatine fossa.

EDITOR'S ABSTRACT.

IODINE THERAPY IN OTORHINOLARYNGOLOGY. JOSEPH BERBERICH, München. med. Wchnschr. **83**:1678 (Oct. 9) 1936.

Berberich introduced the use of strontium salts for the roentgenologic visualization of arteries and veins. He found that strontium can supplant calcium as a therapeutic agent in many conditions, and its use avoids the unpleasant side actions following the intravenous administration of calcium, such as sensations of heat, malaise and vomiting. He then studied chemical combinations of iodine and strontium to be used in place of the various forms of iodine generally used for internal medication. Iodine is used in acute and chronic rhinitis, laryngitis, tracheitis and bronchitis. Because of the unpleasant side actions many physicians do not administer iodine. Berberich observed that strontium counteracts the undesirable effects of iodine as well as calcium does. For that reason he tried a combination of iodine, strontium and silicates in conditions requiring medication with iodine. The results were good, without the development of iodism.

BRAHDY, Mount Vernon, N. Y. [AM. J. DIS. CHILD.]

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Regular Monthly Meeting, March 1, 1937

IRA FRANK, M.D., *President*

WALTER H. THEOBALD, M.D., *Secretary*

OTOLARYNGOLOGICAL DIVISION OF THE ARMY MEDICAL MUSEUM. DR. JOSEPH C. BECK.

Earlier in the evening there was exhibited here a loan set of histopathologic preparations, which was sent to me from the Army Medical Museum in Washington, D. C. I brought them here so that any one who is interested can see what can be obtained.

Two years ago the otolaryngologic division of the registry was started, and since then histologic preparations to illustrate 51 histopathologic processes have been made. Twenty-five sets are available. It is expected that each set will be completed to 100 preparations in due time. Accompanying each set is an atlas showing the gross specimens and indicating just what these slides represent, and there is a written description of each preparation telling just what can be seen under the microscope.

A loan set may be had through the curator of the Army Medical Museum, Major James E. Ash, by writing to him and sending a check for $25 as a deposit. On the return of the set, after three weeks, the money is returned, minus the cost of shipment. If a preparation is broken or lost in the loan period, there will be a charge of $1 per slide.

During the next three months these 25 sets of 51 preparations each will not be available except for the candidates for the next examination by the American Board of Otolaryngology. Three weeks' use of a set with the literature and the time of sending will take about a month for each man. After the examination the sets may be had by any member of the American Academy of Ophthalmology and Otolaryngology, which is sponsoring this section at the Army Medical Museum under the direction of the surgeon general.

I also wish to show a chart which may be obtained, which indicates the type of lesion from which the otolaryngologic division is anxious to get specimens for the museum. Any gross preparation of tissue from the ear, nose or throat will be examined free of charge if it is sent to the museum, and a slide will be returned, with a description of the observations. The division is eager to obtain specimens to illustrate the diseases indicated in the questionnaire, and the chart tells how to send a preparation to Washington so that it will reach there in good condition and may be properly prepared.

It must be remembered that the registry in no wise promises to return the preparation at any definite time and is not in competition with commercial or other business organizations.

Any member of the academy or any otolaryngologist who has a member of the academy sponsor him may borrow these sets.

DISCUSSION

DR. AUSTIN A. HAYDEN: I wish to remind those present that Dr. Beck is largely responsible for the creation of the opportunity which is placed in the hands of over 1,500 otolaryngologists in this country to have slides of pathologic specimens in their own hands for study. I think the otolaryngologists of Chicago should know that he has been instrumental in doing this and that they as well as others will benefit by this effort.

COMMENTS ON TREATMENT OF THE SINUS. DR. ARTHUR W. PROETZ, St. Louis, Mo.

As the first step in the treatment of disease of the sinuses, the normal functions of the nose should be considered. As a second, the deviations from the normal should be evaluated in terms of discomfort and injury to the patient and also in terms of the likelihood of their correction. With this information at hand, it should be possible to select methods which will accomplish most and injure least.

Both inspired and expired air passes through the normal nose in a high, vaulted curve, the inspired air passing largely through the olfactory fissure and the expired air partly through the middle meatus as well. Except for certain eddies, which have been described elsewhere, the currents are much the same.

This is not true, however, of the pressures. The extent of the fluctuation is determined largely by the relative size of the naris and in abnormal noses by the size and location of any obstruction. The greater the obstruction anteriorly, the greater the fluctuation. Naturally, posterior obstructions have the opposite effect. Peak pressures in normal sinuses equal those within the nasal chambers.

Distribution is extremely important. In order properly to temper the inspired air, a large proportion of the mucosa must come into play. If the total volume of air is forced to pass through a restricted channel or channels, these cannot cope with the requirements. The result is overdrying in the exposed areas and overmoistening in the protected ones.

The blanket of mucous secretion is perhaps the most important single factor in the prevention and eradication of nasal infection. It is a thin but tough and viscous secretion. It is entirely renewed once every half hour. This refers to the normal nose; disease conditions impede or even prevent its flow. Any one who has observed living cilia in action must be impressed with the importance of this mechanism. Investigators are agreed that maintenance of the proper moisture and viscosity is vital to its effectiveness.

Cilia are vigorous and resistant and extremely powerful in proportion to their size. Their activity is difficult to suppress; even severe cold (5 C.) only suspends their activity, and they revive on rewarming; they live for hours after death, even though the body is kept in a refrigerator; they persist for days in Locke's solution under the microscope. They are regenerated, together with the epithelium, after complete stripping of a sinus wall; they commonly persist within sinuses in the presence of old and extensive purulent infections.

The one requisite to their existence throughout the animal world is adequate moisture. In the planning of surgical treatment of the nose, therefore, nothing is so important for drainage as avoidance of dry areas.

The exchange of air in a sinus is extremely small, so that little evaporation occurs there and relatively few glands are required to maintain the required moisture. If a large opening is cut into the sinus, however, throwing it into the channel of nasal respiration, drying occurs, with injury or destruction of the cilia. Infectious material continues to be thrown from the remaining walls, and the means of expelling it has been removed.

Contrary to a long-accepted view that nasal infection speedily incapacitates the epithelium and its cilia, these structures are found working vigorously in the presence of pus, even in advanced stages.

The wide-open or half-obliterated cavity receives the impact of inspired air, dries and is unable to cope with its viscous or crusted secretions. A moderate opening, on the contrary, enables the cavity to retain its moisture and encourages its cilia to keep it clean. The various flaps, which are sometimes retained at great pains, do little to remedy this situation in the presence of wide openings, and at the same time they retain much of the infection which lies in their mucosa and might better be eradicated. At least at the time of writing, I believe that an obliterating operation which throws a sinus into the general nasal cavity is to be avoided.

When actual obliteration can be accomplished, with destruction of all walls, as in the frontal sinus, the operation is more likely to be successful. Extensive radical surgical treatment of the sinuses should be regarded as similar to ampu-

tation. There is certainly justification at times for removing a leg, and the operation may be executed with finesse, but the result is a one-legged person from that time on.

DISCUSSION

DR. AUSTIN A. HAYDEN: I should like to ask Dr. Proetz if he will not make some exception to his pronouncement against the use of suction. I presume that he means not to use mass suction. It would seem from some of the pictures that he has shown that suction is possible and possibly of decided advantage if it is not used in a closed cavity and if it is used in such a way as not to injure the mucous membrane—in other words, if it is not massive suction.

As he spoke I was wondering about the care of the mucous membrane in operating, and I noticed that he mentioned the Caldwell-Luc operation at least twice. I presume that a counter opening under the turbinate with the Caldwell-Luc incision is contraindicated, that is, that a second ostium would not be in accordance with his ideas—at least not a large opening that would not promptly close.

I have enjoyed seeing these pictures and know well the difficult photography that was required and the skill that was necessary to get these results. The secretary and the society are to be congratulated on having one of the society's own members, even though he lives in Chicago's largest suburb, come here and present this work.

DR. SAMUEL SALINGER: I happen to have had the good fortune to be present in St. Louis at a meeting of the American Laryngological, Rhinological and Otological Society—about six years ago, I think—when Dr. Proetz presented his first paper on the subject of displacement. At that time he proposed it as a diagnostic procedure. Little did I think then that the subject was to lead to contributions of far greater importance and interest. I have had occasion to read almost every paper written on disease of the sinuses in almost every language in the past four years and have been astonished to note the interest that has been aroused by the work which Dr. Proetz started and to observe the effect it is having on the therapy of sinusitis all over the world. There is a definite tendency to treat the sinus with greater respect and with less surgical intervention and with the use of milder drugs than formerly.

I was pleased to hear Dr. Proetz' remark regarding massive suction, for it has been my habit in the past five or six years to avoid this treatment for the reasons he outlined. If one is to use suction, it seems much better to use a small cannula directly applied into the meatus to withdraw secretions, which will free the ostia in an agreeable manner. Such a method is far more comfortable for the patient and certainly more effective than suction applied at the distant anterior naris.

DR. ARTHUR W. PROETZ: I am glad to be allowed to go into this question of suction a little. By massive suction, of course, I mean taking an olive tip and putting it into the nostril, holding the other nostril shut, and then exerting strong negative pressure, which is a common practice if one can rely on what patients say. I am certainly in favor of using a cannula under direct vision for removing the secretions. If one puts a little ephedrine into the sinus to render the secretion less sticky and lets the cilia remove it, they will do it much better.

As to Dr. Hayden's question about the Caldwell-Luc operation and the counter opening—I make a counter opening. The Caldwell-Luc procedure is quite different from the other things I am talking about. This counter opening has not the disadvantage of throwing the stream of air into the sinus.

CONTRIBUTION TO THE TREATMENT OF VASOMOTOR RHINITIS. DR. ALFRED LEWY.

Caustic applications, surgical treatment and ionization cause symptomatic relief from vasomotor rhinitis, apparently in direct proportion to the severity of the insult to the tissues. For about two years my associates and I have been using resorcinol as an agent for cautery, with a better percentage of success than with

the other treatments mentioned. It is applied as a watery paste over the entire nasal mucosa, including the middle meatus, after a preliminary application of a 3 per cent solution of cocaine. The reaction usually is not severe. Sensitive patients may be given 1½ grains (0.97 Gm.) of pentobarbital sodium. The relief may last from a few weeks to several months, and in a number of patients treated about two years ago there has been no recurrence. It has not been successful in hay fever due to ragweed. Other remedial measures may be used at the same time, e.g., the hypodermic use of free iodine, as suggested by Glasscheib. Bipolar diathermy of the inferior turbinates may be combined with it.

DISCUSSION

DR. IRVING I. MUSKAT: After Dr. Lewy told me about this method, I used it for hyperesthetic rhinitis and had a result similar to his. I think it has been about the best thing I have used, and I have tried the other methods in vogue. I have encountered some untoward reaction in some patients, whether neurotic or not I cannot tell, but physicians know that the hyperesthetic person has some neurotic basis for his condition. The method is good and gives relief, which sometimes lasts for many months, as Dr. Lewy stated.

DR. ALFRED LEWY: I am sorry that I cannot state what the histologic effect is on the mucous membrane. I should like to know about that and shall try to find out.

COOK COUNTY HOSPITAL STAFF CONFERENCE

DR. T. C. GALLOWAY: This presentation is an attempt to show how we conduct our staff conferences at the Cook County Hospital. It is difficult to preserve before such a large group the spirit that has made it seem successful to us. We shall pretend that we are sitting by ourselves and shall try to be as informal and spontaneous as we are at home. We ask that those present remember that victories do not come to autopsy and that criticism of diagnosis and treatment is easy in retrospect, and we ask that he who is without clinical sin shall throw the first brickbat.

Our usual program is to take up: (1) deaths, (2) errors in diagnosis, (3) unusual cases, (4) statistical studies from our ward and (5) reports of special investigations.

ABSCESS OF THE TEMPORAL LOBE. History presented by DR. ROBERT LEWY; pathologic report by DR. BEN W. LICHTENSTEIN.

A. O., a woman aged 36, was admitted to a medical ward in the Cook County Hospital on Dec. 3, 1936. The history obtained was that she had an infection of the ear six months previously, associated with frontal headache which persisted. Two months before her admission to the Cook County Hospital she entered another hospital, and a simple operation on the mastoid was done. The operation failed to relieve her headaches. She was brought to the Cook County Hospital in coma, the exact duration of which could not be ascertained.

Examination of the patient on her admission revealed that the temperature was 100 F., the pulse rate 96, the respiratory rate 22 and the blood pressure 124 systolic and 70 diastolic. The left pupil was fixed and dilated; there were ptosis of the left lid and a suggestion of paralysis of the left facial nerve. The optic disks showed some haziness of the lower margins.

Spinal puncture was done, which showed the fluid to be under increased pressure. The reaction to the Pandy test was 2 plus; 60 cells per cubic millimeter were present; the type was not determined.

A tentative diagnosis of the abscess of the brain of otogenic origin was made and was strengthened by a pulse rate of 52 on one occasion. The patient was transferred to a surgical ward, for it was obvious that immediate surgical treatment was indicated.

Examination in the department of otolaryngology showed a bilateral Kernig sign and a purulent, nonodorous discharge from the left ear. An old mas-

toidectomy scar was noted. The left tympanic membrane was discharging in several places.

The patient was examined by an attending physician, who felt that she had a temporosphenoid abscess on the left side.

At operation a recent antrotomy wound, with considerable softening of the bony structures leading to the tip of the mastoid, was noted. There was organized exudate over the dura of the middle fossa. The dura was greatly thickened. A large temporosphenoid abscess was found with a needle. The lateral sinus and the dura of the posterior fossa were essentially normal. The patient lingered until 10:50 p. m. The temperature rose to 106 F., the pulse became imperceptible, and she died six hours after operation.

Dr. Ben W. Lichtenstein: Autopsy revealed a well nourished Indian woman. On removal of the calvarium and incision of the dura a swollen and edematous brain was seen. The leptomeninges were injected, but there was no apparent leptomeningitis. The defects in the skull and the dura mater, as previously described by Dr. Robert Lewy, were noted. On section of the brain an abscess 5 by 4 by 3 cm. was found in the left middle and the left inferior temporal convolution. This abscess extended from the subcortical white substance into the gray substance, reaching the pia mater in one place, and it was filled with light yellow purulent material. Microscopically the abscess of the brain had a poorly developed capsule. The inner layer consisted of masses of polymorphonuclear leukocytes and cellular débris. The middle layer was rich in fat-laden mononuclear cells and newly formed capillaries, while the outer layer contained large numbers of fibrocytes, plasma cells and gitter cells. The adventitial spaces of the cerebral blood vessels in the vicinity of the abscess were crowded with lymphocytes, presenting the picture of so-called nonsuppurative encephalitis. The leptomeninges at the base of the brain were infiltrated with large numbers of lymphocytes, presenting the so-called aseptic meningitis.

The anatomic diagnosis was: large abscess of the temporal lobe on the left side of the brain, with nonsuppurative encephalitis in the vicinity and aseptic meningitis at the base.

Dr. T. C. Galloway: Can you give the age of the abscess?

Dr. B. W. Lichtenstein: The development of abscess of the brain is a controversial subject, especially its development from a suppurative focus in the middle ear. It is the consensus that there first develops localized pachymeningitis, which spreads inward, provoking localized sealed-off leptomeningitis, and thereby organisms may enter the brain. There are many other conceptions. It may be retrograde through the veins. It is not lymphatic, for the drainage of the fluids of the brain is outward into the subarachnoid space. It is difficult to tell the age of an abscess if the capsule is poorly developed. When the wall is composed of dense fibrous tissue, one can definitely state that the abscess is an old one, but as an abscess of the brain usually grows by the development of foci of suppurative encephalitis in its vicinity followed by disintegration of the primary capsule, the abscess may be of long duration and its wall poorly developed. It may be present for months, but at autopsy much of the abscess wall that had been laid down may have been destroyed by the spreading of suppurative encephalitis.

Dr. Samuel J. Pearlman: It is difficult to make a diagnosis on the entrance to the hospital of a patient in a state of coma when no relative or friend is present to give the history. When, however, there are a history of suppuration in the temporal bone and a further history of an operation on the mastoid some time previously, with severe headache ever since the operation and then a descent into a state of coma, there is present a sequence of events which makes the diagnosis of intracranial suppuration by extension from the middle ear and the mastoid tracts rather plain.

The course to pursue in such a case seems always to be a matter for much debate. Certainly, with a patient so profoundly ill one cannot expect much from surgical treatment, and yet there are cases on record in which a comatose patient

had an abscess of the brain evacuated and made either temporary or complete recovery. Even when surgical intervention seems clearly indicated, localization of the abscess is usually not easy. One may feel that it is located in the temporosphenoid lobe only to find at postmortem examination that it was in the cerebellum. The classic signs of abscess in either location are many times absent in practice. If the surgeon feels that an abscess of the brain is present and does not find it in the most likely location, he should search in the other locality. In this case we felt that we should search the temporal lobe first, but we were convinced that if we did not find it we should then search the cerebellum. As has just been stated, the abscess was found in the temporal lobe, but to no effect. We were distressed that the patient went for two months with headache and perhaps without the benefit of medical advice. An earlier exploration might have done her some good.

As to methods of treatment: I must confess that most of the time I have no positive guide, and authorities differ greatly on the treatment of abscess of the brain. My own feeling has been that the surgeon in most cases is compelled to operate about the time that a diagnosis is made. The patient with uncomplicated abscess and in good general condition should wait as long as possible. Certainly, the figures show that those operated on after the third or the fourth week from the onset of symptoms show a higher percentage of recoveries than those for whom intervention is necessary earlier. However, in most cases the patient comes to us not with an uncomplicated abscess of the brain, but with one associated with some other lesion, such as generalized leptomeningitis, rupture into the ventricle, thrombosis of the lateral sinuses or sepsis. The addition of any one of these complications makes the prognosis for recovery much worse.

As to the formation of a capsule: Again, there is much difference of opinion. Walling off seems to be a process in which time is not the only factor. Some brains seem to show little capacity for walling off. The nature of the offending organism may be important. There are some who feel that they can tell what is going on by spinal puncture. If the number of cells is not high, if most of them are lymphocytes rather than polymorphonuclear cells or if bacteria are absent, then one has reason to believe that the brain is putting up a good fight or that the invading organism is not as virulent as it might be.

In other instances, it is apparent that if an operation is not undertaken at once the patient will have no chance at all. The better the patient looks and feels, in general, the better is the prognosis. Advancing cerebritis without walling off can be guessed at clinically if the patient looks toxic, has a low grade fever and has a high cell count in the spinal fluid, with many polymorphonuclear lymphocytes. While walling off is what the surgeon desires, an abscess of the brain too well walled off offers resistance against cure, for abscess heals by collapse. A thick capsule prevents collapse and hence delays or prevents complete healing. Such abscesses of the brain are best handled by complete enucleation, a formidable procedure in itself. No doubt, too, many patients die because of the eagerness of the surgeon to drain well. It is these repeated attempts at drainage, producing new traumatism, that ultimately produce death.

I should like to see reports on the cure of the abscess of the brain similar to the reports which are made today on the cure of carcinoma, that is, after five years and not after from six months to one year. The figures, I am certain, would be different from those ordinarily seen in the literature. Many patients, apparently cured at the end of from six months to a year, would return for another tapping and so on until finally in one recurrence death would take place. Others would acquire epilepsy, which would make them hopeless invalids, useless to themselves and to society.

To sum up, with few exceptions, the treatment of the abscess of the brain today is not a surgical or a medical triumph. I cannot but feel that physicians are far from a satisfactory understanding of the handling of this disease.

DR. ALFRED LEWY: What was the immediate cause of death in this case of abscess of the temporal lobe, and in what way did it cause the death of the patient?

DR. T. C. GALLOWAY: Perhaps Dr. Lichtenstein can answer that question.

DR. B. W. LICHTENSTEIN: The question as to the immediate cause of the death of the patient is frequently asked of the pathologist by the clinician. In the majority of cases the patient dies from a summation of factors, no one thing in itself being of vital importance. In our case the edema of the brain, the toxic degeneration of its ganglion cells, the inflammation of the meninges and the shock of operation all together played a rôle in provoking the patient's death.

DR. T. C. GALLOWAY: Does Dr. Muskat wish to add anything?

DR. IRVING I. MUSKAT: I have nothing to add, as Dr. Pearlman reviewed the subject beautifully, except that I might talk about the mode of operation in these cases. Physicians know that anesthetics have a deleterious effect on the tissue of the brain and that any overdose of anesthetic increases the encephalitis, and it stands to reason that the smaller the amount of encephalitis that is produced the greater will be the benefit. Ether is the anesthetic least injurious to the brain tissue. Nitrous oxide is bad, for it increases cerebral hyperemia and edema and naturally, therefore, produces more encephalitis. When a patient is comatose and one can use a local anesthetic or no anesthetic, the patient is better off. No antiseptic should be used in the brain cavity, for everything physicians are accustomed to use produces more encephalitis. That does not hold true of the dura, for it will stand chemical trauma. Interference with tissues and the amount of trauma produced by the mallet and gouge should be considered. In irrigation of an abscess cavity physiologic solution of sodium chloride is the only chemical that the brain tissue can tolerate with impunity.

DR. T. C. GALLOWAY: Dr. Pearlman and I have had some debate about the time of operation for abscess of the brain. It is my opinion that almost any patient with an abscess who has to be operated on in the first two weeks will not recover. There has never been a series that equals Macewen's, and all his patients were operated on later. I have a feeling that if one can date the onset of the abscess of the brain by chilliness or other symptoms, one should wait for four weeks, until there is some walling off. It is hard to date the beginning of the abscess in this case, and it is true that in any case this patient would have died.

I should like to ask Dr. Pearlman if he thinks a ventricular puncture would have tided this patient over, or if he thinks the patient should have been turned over to a neurosurgeon?

DR. S. J. PEARLMAN: Speaking of Macewen's figures, it is true that in the past forty years no one has equaled his successes. He had much experience and shrewd common sense and some apparent disadvantages, which really worked in his favor. Changes in the spinal fluid were not stressed as much and hence were not used in his day to indicate operation. Macewen's cases were those of chronic involvement. All the other patients, including those who had complications were dead. Those who lived long enough to get into his hands presented the most favorable condition, and with his simple application of "masterly inactivity," a majority did exceedingly well.

In favor of operation on the mastoid process would be the fact that one would not overlook extradural or subdural collections of pus. From time to time a patient presents the signs of abscess of the brain when he has none. Cleaning out of the suppurative focus in the temporal bone cures many of these. The finding of accumulations of serous fluid and their drainage cure other types of abscess of the brain but there is no simple rule which tells exactly how long to wait.

At one of our conferences Dr. Lichtenstein stated that one should wait as long as possible and that if the time from the possible beginning of abscess of the brain is short, it is better not to search for it but to do a wide decompression, thus relieving the compressive phenomena.

Some persons believe in doing ventricular puncture and so reducing intracranial pressure. Abscesses of the temporal lobe have a tendency to spread toward the ventricle, and such a procedure might hasten a rupture into it. As to whether one should turn patients with abscess of the brain over to a neurosurgeon, I am quite willing to agree that their method of approach offers some advan-

tages, but I am not convinced that in general their results are better than those obtained by otologists, who at least clean out the original source of infection and find a pathway often but are trained to tap the abscess through an infected field. The neurosurgeon has the advantage of attacking through a clean field and requires the otologist to work independently on the suppurative focus in the temporal bone.

DR. J. LIFSCHUTZ: Dr. Curry recently observed a case of abscess of the temporal lobe, in which the abscess was diagnosed and drained; during the operation, although the heart was beating normally, the patient stopped breathing and died, despite artificial respiration for three hours. I had a similar experience in which the patient was kept alive for eighteen hours in a Drinker respirator but finally died. Unquestionably, death was due to involvement of the respiratory center in the medulla. Is there any procedure that can be suggested that would enable the patient under such circumstances to resume normal respiration?

LARYNGITIS (STREPTOCOCCIC). History read by DR. ROBERT LEWY.

J. S., a boy aged 3 years, was admitted to the contagious disease ward of the Cook County Hospital on January 2, 1937, with a high fever and a brilliant rash of two days' duration.

On his admission to the hospital the temperature was 104 F. and the pulse rate 145. The left ear-drum was red and bulging. The tonsils were large, extending to the midline, and were covered with a necrotic black slough. There was a small necrotic area in the center of the hard palate. Inspection of the skin showed a diffuse erythematous punctate rash, with circumscribed pallor. There was marked cervical adenopathy.

A diagnosis of scarlatina was made, with diphtheria to be ruled out.

The patient was given 30 cc. of convalescent serum, after which the temperature dropped from 104 to 99.8 F. It was felt that he had a secondary Vincent's infection. The condition continued to run a stormy course. On January 5, 30 cc. more of serum was given; 285 cc. of titrated blood and 3,000 units of diphtheria antitoxin also were administered.

On January 8 it was felt that the child was improving. The leukocyte count was 26,000, and the differential count was normal. On January 10 stridor developed and he was examined by an otolaryngologist. This was the eleventh day of the attack of scarlet fever and the sixth day after the onset of a condition suspected of being diphtheria. Examination showed supraclavicular retraction and an extensive membrane over both tonsils and the posterior part of the pharynx. An exudate from both arytenoid cartilages was noted; the vocal cords were not observed. There was some edema of the epiglottis.

The child was carefully watched. The stridor increased during the night. A tracheotomy was considered, but intubation (experimental) was done first. This relieved the stridor. He was given another transfusion of 285 cc. of blood, and on January 19 he received 250 cc. of blood. The temperature was dropping. Extubation and reintubation were done several times, the last on January 23.

Other complications included bilateral otitis media, which persisted in the left ear for eight weeks and in the right ear for six weeks. The condition ran a septic course for two weeks, with the temperature up to 102 F. daily, and a mastoidectomy was done on the left ear, with relief of symptoms.

DR. T. C. GALLOWAY: Dr. Curry, will you tell us about this case?

DR. L. T. CURRY: This case presented several problems, but there were two which I consider important. First, when should the asphyxiation be relieved? Stridor and retraction of the supraclavicular spaces are my indications for the necessity of promptly relieving the asphyxiation. This child was thin, with marked stridor and marked supraclavicular retraction. It is the degree of these two symptoms, especially in a fat child, which makes difficult the decision of when to intervene.

The other problem was whether to do an intubation or a tracheotomy. I think most otolaryngologists feel that tracheotomy is indicated, but that is because they

do not have a Dr. Hoyne to do the intubation. Intubation worked well in his child, but when Dr. Hoyne is not present, it is a problem to have an assistant who is capable of replacing the intubation tube. I think it is necessary always to have an expert in this line on the job. Unsuccessful intubation makes for a graver prognosis because dypsnea leads to a fatal outcome if it is not relieved promptly.

DR. T. C. GALLOWAY: One of the best things about our staff meetings is that through them we come in contact with the other departments. We are glad to have Dr. Hoyne, from the Contagious Disease Hospital of Cook County, come to our staff meeting and talk about this particular case.

DR. ARCHIBALD HOYNE: The honor conferred on me by my being permitted to talk here is greatly appreciated, but I feel considerable timidity in undertaking to refer either to tracheotomy or to intubation before a gathering of this kind.

The history just given is excellent. I think that ordinarily such a patient requires tracheotomy. When O'Dwyer devised his instruments for intubation, he expressed the belief that his operation might be used for practically any condition in which tracheotomy is indicated. Few will claim that at present. Tracheotomy may be resorted to for any condition in which intubation would be adequate, but intubation may not afford relief in many conditions in which tracheotomy is successful. For a while this particular patient afforded a good topic for debate on the subject of tracheotomy and intubation, and his case was one of those in which properly, perhaps, tracheotomy should have been done. For a long time—and I have not changed my feeling much—it has seemed to me that a patient with diphtheria who cannot be saved by intubation will not be saved by tracheotomy; yet that is not inevitably true. Occasionally a patient with diphtheria may have so much membrane above the glottis, as sometimes happens in the "bull neck type," that it is necessary to do a tracheotomy. However, in a case of tracheobronchial involvement, if intubation will not save the patient tracheotomy rarely will, for there is obstruction below the bifurcation. Nevertheless, in such cases my associates and I always do an intubation first, and if that fails, as it usually does, then we do a tracheotomy—but that nearly always fails too. In streptococcic infections with marked edema of the throat, such as may occur in scarlet fever, measles and a variety of other infectious diseases, usually tracheotomy must be given preference rather than intubation. Nevertheless, if the patient comes to the Contagious Disease Hospital, usually an intubation is done first. There is no objection to inserting a tube, even though it does not accomplish the intended purpose.

In this particular case I was much surprised to find that the child was better after intubation. The edema did not well up over the top of the tube, although the tube did sink deeply into the larynx. The next day the tissues were swollen to such an extent that it was necessary to remove the tube and put in a larger one, and I thought a tracheotomy might not be avoided. Possibly the results would have been better if there had been a tracheotomy. We prefer that members of the department of otolaryngology do our tracheotomies, and their cooperation is appreciated.

Recently Chamberlain published an article on tracheotomy and recommended using the Mosher tube. We have been relying on that tube when performing tracheotomy at the Contagious Disease Hospital for about fifteen years. I think I was responsible for buying the tubes and suggesting their value in tracheotomy. With the tube in place, time is not such an important factor in the operation.

It seems to me that in diphtheria intubation should be done in preference to tracheotomy if the patient is in a hospital in which there is always some one present who can intubate. If the child is at home or in a hospital for the care of patients with noncontagious diseases where there is no one with experience in intubation, tracheotomy is far safer. Particularly is this true if the operator is well qualified. Rarely, cases are reported in which, with too much enthusiasm, "the surgeon" penetrates not only the trachea but also the esophagus, and I imagine that it is not good for the patient. Of course, in connection with intubation

errors occur that are just as strange. When a novice inserts a tube in the esophagus instead of in the larynx and the patient dies, he is likely to regard it as a peculiar case. A patient was brought in one time with a piece of thread hanging out of the mouth. He had been rushed to the hospital for intubation. Seeing the string I asked if a tube had been put in the throat. The mother said: "Oh yes, he has two tubes." He had two, but he needed a third—the other two were in the stomach.

This subject of intubation and tracheotomy is a matter on which the staff members of the department of otolaryngology and of the department of contagious disease of the Cook County Hospital are fairly agreed. We are all of the opinion that one should try to do what is best for the patient, irrespective of one's personal preferences in the choice of operative procedure.

DR. T. C. GALLOWAY: I think I should mention the possibility of direct intubation, which gives a chance to remove membrane. We should recognize that Dr. Hoyne is without a peer as an intubationist, and if I had diphtheria and the question of intubation came up I am sure I should want Dr. Hoyne to do the intubation. I think that we of the department of otolaryngology feel strongly that in tracheobronchitis it is necessary to have a tracheotomy for proper removal of secretion and crusts.

TUBERCULOSIS OF THE TRACHEA WITH SYPHILITIC ENDOCARDITIS. History presented by DR. ROBERT LEWY; pathologic report by DR. BEN W. LICHTENSTEIN.

A. H., a white woman aged 33, was first admitted to the Cook County Hospital in March 1936, with a tentative diagnosis of toxic nodular goiter. It was learned that she had noted an enlargement of the thyroid for eight years and had lost about 45 pounds (20.3 Kg.). During the past year she had been nervous and had marked weakness of the left arm. The basal metabolic rate was +12, +9 and +2, respectively, on three occasions. Careful investigation excluded the possibility that the patient had a toxic goiter.

Physical examination showed an absence of the radial and brachial pulsations in the left arm. The blood pressure in the right arm was 120 systolic and 70 diastolic; it was not obtainable for the left arm. It was thought that the patient might have a cervical rib that was compressing the subclavian artery, but roentgenographic investigation gave negative results.

Laryngeal examination on April 4 showed that both vocal cords moved normally. No tumor was seen.

Operation was performed the following week. Obliteration of the second portion of the subclavian artery was found. The artery seemed to be surrounded by dense connective tissue. It was cleared, and a segment was excised. The patient made an uneventful recovery and left the hospital after four weeks, her condition not much improved.

Four months later the patient was readmitted to the hospital because of marked dypsnea, which was particularly noted when she lay down and after exertion. At that time it was learned that her husband was being treated for tuberculosis. Physical examination showed that she was having labored respiration with a wheezing noise and was cyanotic and dypsneic. The heart and lungs were essentially normal. The leukocyte count was 2,150.

At that time a diagnosis of tracheal obstruction was made, and the patient was sent to the otolaryngologic department for bronchoscopy. A mass about the size of a marble was seen bulging into the lumen of the trachea. This was above the carina near the bifurcation, about 30 cm. from the incisor teeth, and it obstructed about three fourths of the lumen.

A specimen of the mass was taken for biopsy because it was thought to be an inflamed gland. The pathologic diagnosis was fibrocaseous tuberculosis.

Bronchoscopy was again done on September 12, and removal of the mass by desiccation and diathermy was suggested. A low tracheotomy also was advised. Roentgen therapy was given, with definite temporary improvement. A roent-

genogram of the chest at that time showed no mediastinal mass and no evidence of tuberculosis or cervical ribs.

The patient's legs became edematous; a low grade fever developed, and she coughed a great deal and had much difficulty in breathing. She died three months after her second admission to the hospital.

Postmortem examination of the blood showed the Wassermann reaction to be 4 plus and the Kahn reaction positive.

DR. B. W. LICHTENSTEIN: Autopsy was performed by Dr. R. H. Jaffé. On removal of the manubrium of the sternum the anterior part of the mediastinum was seen to be bound down by fibrous adhesions. The arch of the aorta was the site of marked wrinkling and puckering of the intima, characteristics of syphilitic aortitis. These syphilitic changes had completely occluded the ostium of the left subclavian artery. The essential pathologic involvement was found in the trachea. When it was opened posteriorly, an ulcer 5 by 3 cm. was found just above its bifurcation on the right lateral wall. This ulcer extended for a short distance into the right main bronchus; its floor was necrotic, and several rings of the trachea were exposed. Adherent to the trachea at this point were several enlarged lymph glands, which on section were composed of dry yellow caseous material. Microscopic examination showed the glands to be tuberculous. In the lower lobe of the right lung a small subpleural nodule was found, which was interpreted as the residue of the primary infection. A chain of enlarged caseous tuberculous lymph nodes extended up along the right main bronchus to the region of the bifurcation of the trachea.

The anatomic diagnosis was: (1) caseous tuberculous lymphadenitis of the peribronchial and peritracheal lymph nodes, with perforation of one of the lymph nodes into the trachea and the production of a tuberculous ulcer; (2) marked syphilitic aortitis in the arch, with complete occlusion of the ostium of the left subclavian artery.

Pathologically several types of tuberculosis of the trachea are differentiated: first, the type associated with massive pulmonary tuberculosis and tuberculosis of the larynx; second, the type that results from the development of a tubercle in the mucosa from lymphatic spread of the tuberculosis in the lungs; third, tuberculosis of the trachea as part of a miliary spread; fourth, caseous tuberculous peritracheal lymphadenitis, with erosion through the wall of the trachea and the production of a tuberculous ulcer and finally of primary tuberculous infection in the trachea.

Our case represents that form of tracheal tuberculosis which is a complication of an endoglandular exacerbation of tuberculous lymph nodes.

DR. ALFRED LEWY: This rare case presents two interesting features. First, clinically and roentgenologically no pulmonary tuberculosis was demonstrable during the life of the patient. The diagnosis was made when the tracheal lesion caused enough dypsnea to have the patient transferred to the department of otolaryngology, where bronchoscopy was done, a biopsy made and the lesion diagnosed. It is evident that a careful autopsy is necessary to exclude the possibility of an old tuberculous lesion which may have caused primary laryngeal tuberculosis, as is shown by this case of tracheal tuberculosis.

The other interesting thing is that at the Cook County Hospital, where Wassermann and Kahn tests are a part of the routine of examination, so far as the record shows, no such tests were made and the syphilis went undiagnosed until the patient came to the autopsy table, where the postmortem Wassermann test revealed it.

Book Reviews

Les maladies du pharynx. By Prof. G. Canuyt (Strasbourg), with the collaboration of Professors Reverchon (Lille), Truffert (Paris) and Wild (Strasbourg). Pp. 790, with 216 figures. Paris: Masson et Cie, 1936.

This excellent treatise is divided into four parts, concerning the oropharynx excluding the tongue, the nasopharynx, the hypopharnyx including the upper aspects of the larynx, and medicolegal aspects of disease affecting this region. The anatomic structure of the peritonsillar region and of the peripharyngeal spaces is accurately depicted in original diagrams. Dental infection is included in considerable detail. The pathologic picture of malignant growths is well illustrated and discussed, and the differential diagnosis of tuberculosis and syphilis is shown by good color plates. Reverchon contributes good chapters on paresthesias and paralyses, and two Italian authors discuss tonsillitis in relation to the kidneys and to rheumatism. The discussion of septicemia is valuable, though it does not include sulfonamide therapy. Truffert's chapters on laryngopharyngeal cancer and benign tumor are admirably clear. The discussion of cancer of the nasopharynx is extensive, carefully documented and well illustrated. While there is no subject index, most of the chapters are logically arranged and followed by comprehensive bibliographies, mainly from Continental and English authorities. Canuyt has an amusing reference to early enthusiasm over the theory of focal infection in the United States and abroad, counseling circumspection with the shrewdness and clarity characteristic of this excellent teacher, who owes his fine clinic to the generosity of the Rockefeller Foundation.

Directory of Otolaryngologic Societies *

FOREIGN

COLLEGIUM OTO-RHINO-LARYNGOLOGICUM AMICITIÆ SACRUM

President: Prof. Gunnar Holmgren, Karolinska Mediko-Kirurgiska Institutet, Stockholm, Sweden.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg, 143 Groningen, The Netherlands.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Leroy A. Schall, 270 Commonwealth Ave., Boston.
Place: San Francisco.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Frank E. Burch, 408 Peter St., St. Paul.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Palmer House, Chicago. Time: Oct. 10-15, 1937.

AMERICAN BRONCHOSCOPIC SOCIETY

President: Dr. Joseph C. Beck, 185 N. Wabash Ave., Chicago.
Secretary: Dr. Lyman Richards, 319 Longwood Ave., Boston.
Place: Atlantic City, N. J. Time: June 2, 1937.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. W. B. Chamberlain, 10515 Carnegie Ave., Cleveland.
Secretary: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Place: Atlantic City, N. J. Time: May 31 and June 1-2, 1937.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Hotel Traymore, Atlantic City, N. J. Time: June 3, 4 and 5, 1937.

SECTIONS:

Eastern.—Chairman: Dr. D. C. Jarvis, Quarry Bank Bldg., Barre, Vt.
Southern.—Chairman: Dr. Charles D. Blassingame, P. & S. Bldg., Memphis, Tenn.
Middle.—Chairman: Dr. Alfred Lewy, 25 E. Washington St., Chicago.
Mid-Western: Dr. Frederick A. Figi, 436 Tenth Ave., Rochester, Minn.
Western.—Chairman: Dr. David R. Higbee, 3245 Fourth Ave., San Diego, Calif.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. Edmund Prince Fowler, 140 E. 54th St., New York.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.
Place: Lido Club, Long Beach, N. Y.: Time: May 27-28, 1937.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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